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FAMILIAL CATARACT WITH EXTENSIVE PEDIGREE CHART*†

BY

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THE purpose of this paper is to present an interesting and hitherto unreported genealogy of familial cataract, to describe the biomicroscopic features of the cataract and to discuss a few published papers in comparison with the case herein reported. The cataract is not present at birth but has been observed as early as six years and may be as late as thirty, forty or even fifty years of age in showing itself by causing serious loss of vision. Cases of early and late development co-exist so that it is impossible to say that the cataract shows the characteristic of anticipation. Its features are so characteristic that a member of the family possessing the cataract may be identified with certainty on examination of the eyes.

* A paper read before the Midland Ophthalmological Society at Dudley on April 6, 1945, with a few subsequent additions.

† Received for publication, February 4, 1947.

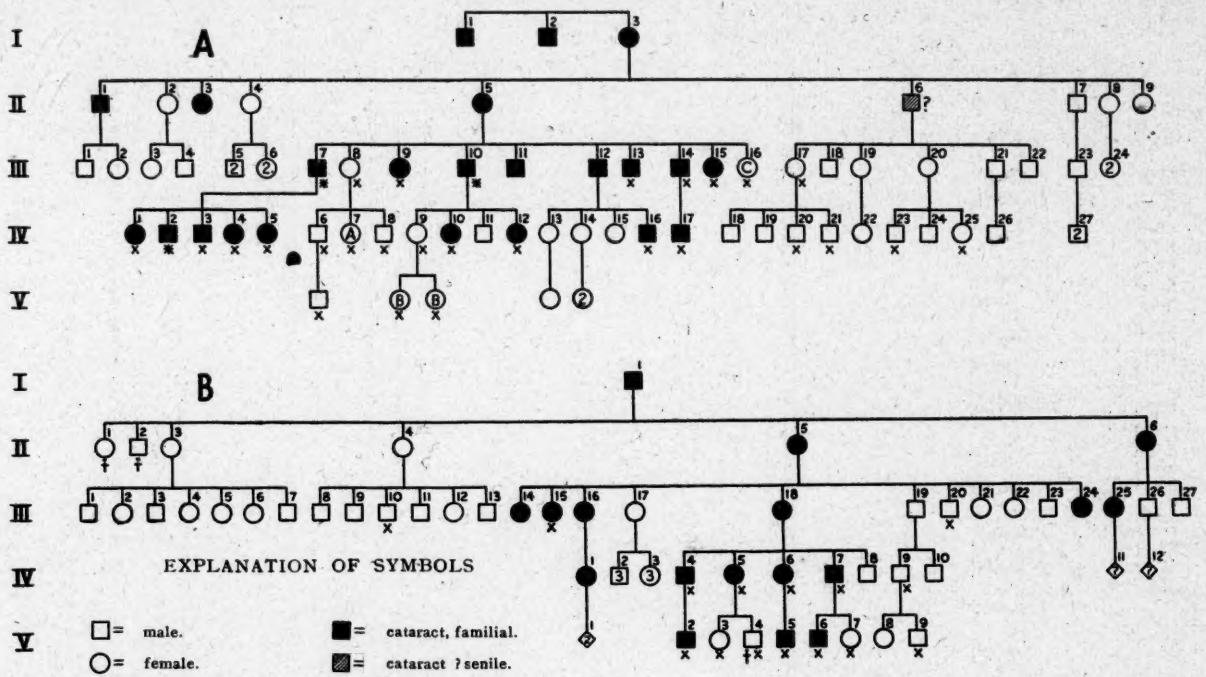
A
B
EXPLANATION OF SYMBOLS
□= male.
○= female.
2= 2 siblings, male.
2= 2 siblings, sex unknown.
?= number unknown, sex unknown.
x = examined by author.
* = examined by other competent observer.
= cataract, familial.
= cataract ? senile.
A= blue sclerotics and hare lip.
B= maculo-cerebral degeneration.
C= retinopathy.
† = died in infancy.

FIG. 1.

The study of the genealogy, extending over the last eleven years, has provided a great deal of human interest. My attention was first drawn to one branch of the family (Fig. 1 A) by the late Bernard Cridland at the Wolverhampton Eye Infirmary where one of the senior members of the family, Miss Clara Attwood (AIII 9), was operated on by him in 1935. Miss Attwood's intimate knowledge of the family history has been of great assistance in preparing the genealogy of this branch of the family. The existence of the other branch was discovered in a rather dramatic way. While several members of the Attwood family were being examined with the slit-lamp, a member of the other branch (J. E. L., B. IV. 4) came into the Out-patient department for removal of a foreign body from the cornea. The nurse on duty, on the watch for such a combination of circumstances, announced that she had a *young* man with *cataract*. Examination with the slit-lamp made it quite plain that he belonged to the family with this particular and distinctive cataract. A common ancestor could not be established at once, nor could any relations of the same name. Considerable research by J. E. L. eventually established the link. His great-grandfather (B. I. 1) was named Elwell (born 1807). The great-grandmother of generation A.IV. was also an Elwell (born 1815). It is probable that they were cousins but this point has not yet been cleared.

These Elwells of a century ago lived at Sedgley, between Dudley and Wolverhampton. Their descendants are now scattered widely over the Midlands (Wolverhampton, Walsall, Birmingham, Coventry and Northampton), some are in North Wales, some in Monmouthshire, some in London, some in Manchester. Two branches of the family are in the United States of America (Rhode Island) and at least one member migrated to Australia. Many still live in the Dudley-Sedgley area. They are a healthy stock, fairly prolific, and, apart from the peculiar cataract which is the subject of this paper, free from physical and developmental defects. They would rank above average in physical and mental equipment. No consanguinity has been discovered in the genealogy. There is no evidence of calcium deficiency and the blood chemistry and Wassermann reaction are normal in those members of two generations who have been examined. (See note later in this paper for details and exceptions).

Fig. 1.

* = examined by other competent observer.

Description of the cataract

Distinctive features of the cataract are (*a*) its presenile onset, (*b*) its position, primarily posterior, sub-capsular, and therefore saucer-shaped with concavity forwards, thus somewhat resembling cataracta aparathyroidea and ray cataract, (*c*) its configuration, leaf

or feather-like branches radiating from the centre, thus somewhat like concussion cataract, and (*d*) flakes and dots in white, yellow, green or blue, throughout the cortex but always separated from the anterior capsule by a sub-capsular clear zone, the zone of disjunction, (*e*) accentuation of the shagreen of the anterior capsule. The earliest signs observed so far are visible only with the slit-lamp.

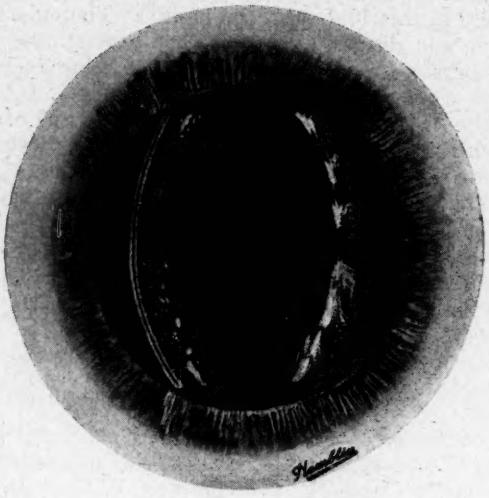

FIG. 4.

October, 1935. The narrow beam appearance of the same case as Fig. 3.

The posterior cortex gives a whitish reflex similar to, but less marked than, that found in the later stage. The difference is that no opacity can be detected by ophthalmoscopic examination.

The illustrations, Figs. 2, 3 and 4, will amplify this brief description.

Analysis of the pedigree

Fig. 1 has been divided into two parts, A and B, partly because the connection between the two families has not been established, and partly for convenience. Part A consists of 73 persons, 36 male and 37 female. Of the 73 there are 23 affected, 13 male and 10 female. Part B includes 58 persons, 28 male and 28 female, while the sex of two has not been ascertained. Of the 58, the number known to be affected is 17 (6 male, 11 female).

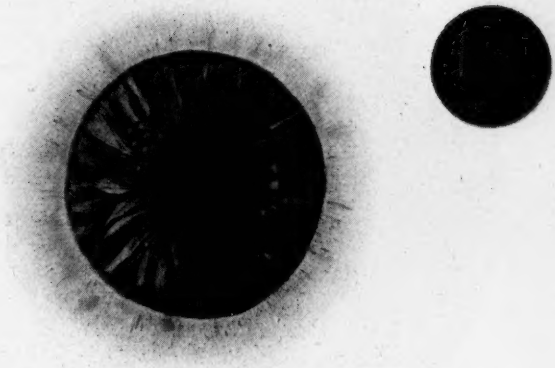

FIG. 2 (A.III. 14).

October 1935. The cataract is well advanced. The small picture shows the appearance by distant direct ophthalmoscopy. The larger one shows a composite slit-lamp appearance The denser large opacities are posterior and curved like a saucer The dots are scattered throughout the lens cortex.

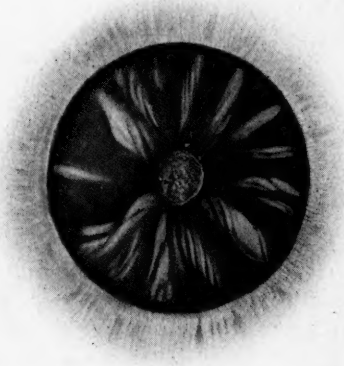

FIG. 3 (A.IV. 17).

October, 1935. This cataract is much less advanced than that in Fig. 2. The central opacity is in the deepest layers of lens cortex, in the position shown by the interrupted white line in Fig. 4. The radiating spokes are at the same depth.

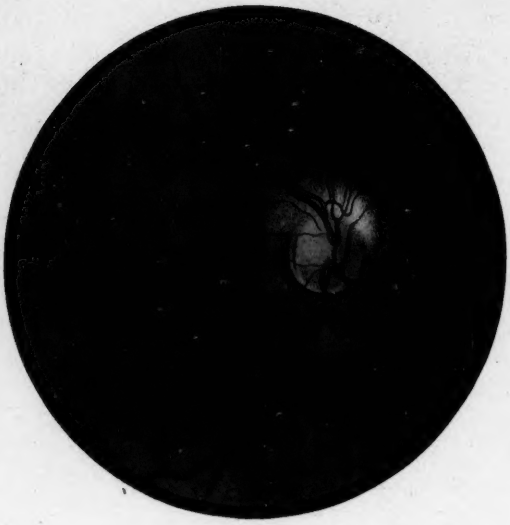

FIG. 5. (A.III. 16).

October, 1935. A peculiar pitted appearance of the central area of the retina. The round marks appear like depressions and have no resemblance to colloid or other forms of degeneration.

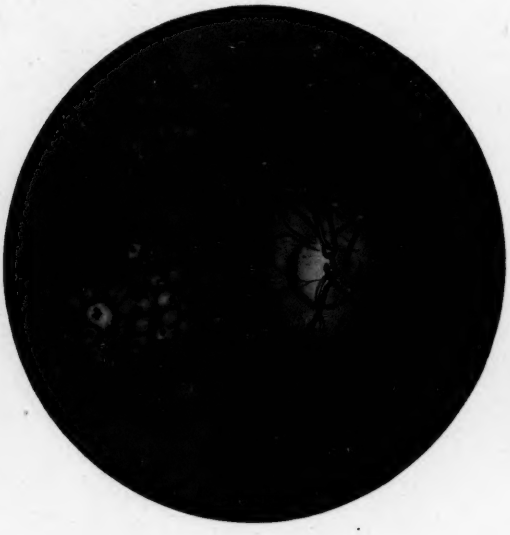

FIG. 6.

The same eye as Fig. 5, but the drawing was made in June, 1945, when the appearance was that of "Doyne's honeycomb choroiditis."

The affected persons in Part A are 31·5 per cent. of the total, in Part B 29·3 per cent. In Part A there is a slight preponderance of males among the affected members; in Part B the affected females are nearly twice as numerous as affected males. There is no particular significance in these relative figures. What is significant is that inheritance is through either sex and through affected members only. It is therefore a Mendelian dominant characteristic. Forty-one members of the family have been examined by the writer, the majority in the years 1935 and 1936. Several have been seen periodically during the last ten years and a number of these have had one or both cataracts removed during that time.

The members of the family may now be dealt with seriatim, beginning with Part A.

A.I. 1-3. Michael and Joseph Elwell were known to have had cataracts removed. Sarah (Elwell) Hedges knew that it was hereditary. She also had her cataracts removed. She was born in 1815 and died in 1892 and is well remembered by her living grandchildren.

II. 1 and III. 1, 2. Charles. Had operation. Migrated to America. Whether his two children were affected and whether they have children is not known at present.

II. 2 and III. 3, 4. Martha—unaffected and her children unaffected so far as symptoms go (not examined).

II. 3. Mary. Unmarried. Cataracts removed.

II. 4 and III. 5, 6. Unaffected and living in America.

II. 5. Phoebe Attwood (née Hedges). Cataracts removed. Was perturbed because her children were affected. Some remained single for that reason. Of her ten children eight are affected.

II. 6. David. Died nearly 70 years of age. His doctor said he had cataract but he did not have an operation. Possibly his was senile cataract. None of his children or grand-children is known to be affected. Five have been examined by the writer.

II. 7. Harry. Not affected, nor are his son or two grandsons (not examined).

II. 8. and III. 24. Unaffected and living in America (Rhode Island).

II. 9. Sarah. Unaffected. Did not marry.

III. 7. Harry Attwood. Operated. All his five children affected. The two youngest have the earliest signs yet observed, visible only with slit-lamp.

III. 8. Jessie. Unaffected and her children unaffected.

III. 9. Clara. Unmarried. Giddiness on wearing cataract glass after operation. Difficulty disappeared after gall bladder operation.

III. 14. Sidney. Operation by G. F. Haycraft 1936. Was able to work till the cataracts were far advanced (Fig. 2).

III. 16. Ada. Unmarried. Unaffected. Had a very unusual fundus abnormality. In 1935 the central area of the retina had the appearance of numerous small round depressions as if it had been struck by pellets from a shot gun, or they may have been elevations like bubbles. The colour was that of normal retina. (Fig. 5). In April, 1945, the condition had quite altered. It is not unlike Doyne's familial honeycomb choroiditis (Fig. 6).

IV. 3. Frank, aged 32 years. Served in recent war but was discharged on account of failing vision. Left cataract removed in 1943. Right developed rapidly and was removed in 1945.

IV. 4, 5. Florence and Miriam. No ophthalmoscopic sign of cataract but the posterior cortex reflects the slit-lamp beam as effectively as a developed opacity. The glow is white.

IV. 7. Not affected. Has blue sclerotics and hare lip.

IV. 10. Right cataract removed in 1944, left in 1946.

IV. 12. One cataract (? left), removed at Northampton 1942.

IV. 13-15. V. 4, 5. All living at Newport, Mon. and not examined.

IV. 17. George Attwood, aged 27 years. Affected. There has been but slight advance in his cataracts in the last ten years (Figs. 3 and 4).

V. 2, 3. These two children developed maculo-cerebral degeneration and are the subjects of a previous report (Johnstone, 1938). The elder died in February, 1943, aged 18 years. The younger died in December, 1945, aged 19 years.

PART B

B.I. 1. Benjamin Elwell, born 1807. Had one cataract removed and lost the eye. Refused operation on the other eye. His grandson (III. 10), used to lead him about Sedgley streets. His relationship to his contemporary Elwells (A.I. 1-3) has not been established, possibly cousins.

II. 5. Elizabeth Williams (née Elwell). Herself and five of her eleven children affected.

III. 10. Benjamin Russell. Belongs to an unaffected branch. His knowledge of the family has been invaluable in the investigation. He was interviewed in 1938. No attempt was made to chart the unaffected branch of the family any further than his generation as the members were somewhat scattered, many of them dead, and he knew of no cataract among them.

III. 14, 15. Both affected and both died without issue.

III. 16. IV. 1. Cataracts removed at Manchester.

III. 18. Louisa Lowndes (née Williams). Herself and four of her five children affected.

IV. 4. Joseph Lowndes, aged 45 years. Left cataract extracted by Bernard Cridland in 1931, the right by the writer 1942. Passed A.1 for Army in 1916, obtained marksman's proficiency at musketry and went overseas in October, 1916. Became a mustard gas casualty on November 9, 1918, but no after-effects. His interest and co-operation have made the investigation of Part B of the family possible. He is still following every available opportunity to extend his knowledge of the family. He is a skilled workman and foreman in the engineering trade and apparently was not

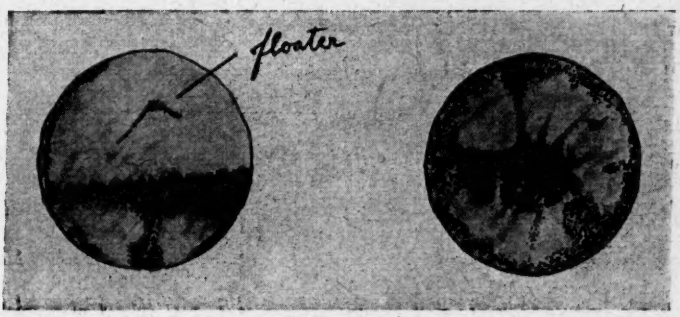

FIG. 7.

Subjective appearance in the eyepiece of a microscope drawn by Joseph Lowndes (B.IV. 4). R. cataract. L. aphakia with some capsule remnants. A vitreous opacity is shown in the upper part of field. Drawn June, 1941, a year before right cataract extension.

handicapped while using one aphakic eye during at least seven years when the right cataract was developing. In 1941 he prepared a drawing showing the shadow of his right cataract seen subjectively when looking into a microscope (Fig. 7). With it is shown the shadow from capsule in his left eye and also a vitreous opacity.

IV. 5. Beatrice. Both eyes operated on in 1918 by Bernard Cridland.

IV. 6. May. Awaiting operation now.

IV. 7. Harry, aged 39 years. Left extraction by Bernard Cridland about 1914, right by the writer in 1945.

V. 2. Dennis, aged 19 years. Both cataracts extracted by the writer.

V. 5. Gordon Stones. 1937 (aged 9 years) no sign of cataract. 1945 early signs present with posterior reflex and a few flakes.

V. 6. Graham, aged 6 years. Examined January, 1945. Left cataract with feathery posterior pattern and many scattered dots. Right less advanced.

V. 7. Viviene (born March 15, 1943). No obvious opacity. Examined without mydriatic and without slit-lamp.

TABLE I

	Date of Test	Serum Calcium	Serum Cholesterol	Serum Phosphorus	Serum Protein		
					Total	Albumin	Globulin
Normal	—	9–11	100–200	2–4	5–8	3–5	1–2·5
A III 9	Sept. 1946	8·1	227	—	—	—	—
A IV 3	Sept. 1945	10·0	not done	not done	7·96	5·67	2·29
A IV 10	Aug. 1946	7·14	262	3·9	—	—	—
A IV 17	April 1946	12·0	80	3·6	8·46	5·86	2·60
B IV 7	June 1945	12·0	65	3·2	7·30	3·41	3·79
B V 2	April 1946	12·0	65	3·6	7·10	5·67	1·43
A. L.	Sept. 1946	10·2	174	2·7	—	—	—
E. G.	July 1946	11·4	174	—	—	—	—

All figures are given in mg. per cent.

In Table I the summary is given of all blood estimations so far carried out. They have all been done in the last two years and the number is still small. The last two cases, A. L. and E. G., have familial cataract but do not live in the Dudley area and their connection with the main pedigree has not been traced. Serum cholesterol varies considerably in the series—two are above, three below and two are within normal limits. In the case of calcium, four are above, two slightly below and two within normal. The serum phosphorus and protein are normal in all the cases in which they were estimated.

Discussion of some published papers

In the first decade of this century, particularly from 1906 to 1910, much interest was shown in the genetics of transmissible eye defects. Nettleship and Ogilvie (1906) published the pedigree

of the Coppock family of Headington Quarry, Oxford. This family and cataract are widely known to ophthalmologists, having been discovered by Doyne in 1888 at the Oxford Eye Hospital. Adams continued Doyne's interest in the family so that few who have taken the course for the Oxford Diploma in Ophthalmology or attended the Oxford Congress, can fail to have some acquaintance with the "Coppock cataract," or "Doyne's discoid cataract." Adams (1942) has brought the story up to date with a description of the slit-lamp appearance. A point of minor interest in Nettleship and Ogilvie's paper (1906) is that the obtaining of the full genealogy of the Coppock family was entrusted to the Vicar of Headington Quarry, Johnstone by name. Doyne's discoid cataract is quite definitely congenital, the opacity involving some part of the lens nucleus and being present at birth. It is transmitted as a Mendelian dominant.

Nettleship (1909) published seven more genealogies of cataract, one of which was congenital (Case 1, Everett), one was senile (Case 4), three were possibly familial (Case 2, Perrin), (Case 3, Tomes), (Case 6, Deasley), and two were very likely familial (Case 5, Hiblen), (Case 7, Oldfield). The same author (1912) published a further case of presenile cataract.

Coming to more recent literature on the subject—Veil and Favoury (1930) present a case of probable Mendelian dominant inheritance. The pedigree is far from complete and the cataract is described as "cataracte nucléo-corticale postérieure nummulaire avec disques stratifiés, entourés d'un halo poussiéreux." Like the subject of this paper, there is accentuation of the anterior capsule shagreen, some dots in the anterior cortex and the main opacity is posterior, but there the similarity ends. The chief opacity is from behind the foetal nucleus into the posterior cortex. It presents a stratified structure in concentric lamellae, indented and fitting into one another, the whole resembling a piece of money centred on the axis of the lens. It is surrounded by a ring of dust-like opacity affecting the posterior capsule, the periphery of which is transparent.

Weill and Nordmann (1930) have collected a series of six cases of endocrine disturbance with lens opacities. They divide them into two groups, (a) posterior saucer-shaped and (b) punctate and flocculent. In the first group there are three cases. Two are due to parathyroid deficiency and agree with this condition. One is a severe case of diabetes, aged 38 years. There are fine dots and some red and green crystals at the anterior surface of disjunction in addition to the posterior "cataracte en soucoupe." The description suggests the picture of the cataract of this paper but the drawings are not like it at all. In group (b) two cases of myotonic

dystrophy present features resembling the present series more nearly. The third case has all the appearance of a lamellar cataract with riders; serum calcium below normal, Chvostek's sign positive and history of convulsions at age two. There is nothing in the genealogy of this present report to compare with the endocrine disorders of the six cases above.

Two papers by Caughey (1933) and Souter (1933) deal with cataract in dystrophia myotonica. The cataract in these cases has much in common with the cataract in this paper but my cases have no suggestion of muscular dystrophy or endocrine disorder. For a fuller appreciation of cataract in myotonic dystrophy the reader is referred to the above papers and to articles by Adie and Greenfield (1923) and Goulden (1928), quoted by these authors.

The purpose in referring to these cases from the literature is to draw attention to the points of similarity between cataracts associated with endocrine and metabolic disorders and my own cases without such disorders. When more is known about genetics and about endocrinology it may be this similarity will be explicable. For the present there seems to me no alternative to the view that the cataract of this paper is an hereditary feature transmitted by an autosome. As such it is not a defect or degenerative tendency on the part of the lens or the endocrine glands, but a true inherited feature. It is not apparent at birth and so cannot be called congenital. It will resemble the affected parent later on if the infant has the autosome bearing the characteristic. It may be compared with a distinctive type of nose, for example. At birth and in infancy the nose is like any other baby's nose, but as the child grows up and approaches maturity the nose develops the parental type and the similarity is easily recognised. We may say it is a familial, an inherited characteristic, but not a congenital one.

There is one more paper to which reference must be made. Hornback and De Garis (1933) give a pedigree made up of 59 persons in four generations of whom 30 had cataract, 14 male and 16 female. The unusual feature is that two males in generation II appear to have transmitted the cataract without themselves being affected. The authors rightly argue that the inheritance is an autosomal one, and dominant. To explain how an unaffected parent could have affected offspring they postulate a dominant autosomal gene which is atypical in that it produces its effect (cataract) only under certain conditions. There seems one fatal weakness in their line of argument. They produce no evidence that the two males in question didn't have cataract, beyond the fact that they were not operated on or left no history of faulty vision. Evidence of absence of cataract in the light of present day knowledge could be accepted only after examination with the slit-lamp

and corneal microscope. From my experience familial cataract may be present for many years without the affected person having any suspicion of it. The age at which it begins to interfere with normal work and activity depends upon whether a clear space remains in or very near the visual axis or not. Some cases have been able to go on working and reading till a marked degree of opacity is visible with the ophthalmoscope; a few are in difficulty when the opacity is comparatively small but more obstructive to direct vision. The cataract is never so visible to the casual observer as a mature senile cataract. A medical practitioner may well be excused for not being able to tell if the cataract is present in the early stages. Ophthalmic surgeons may miss the cause of failing vision in young people, as actually happened with two of the members of the family herein reported. In my opinion the view that some other factors, possibly nutritional or endocrine, predispose to the realisation of the hereditary characteristic (cataract) is not proven. The " carriers " may have had the defect.

Further study of blood chemistry and endocrine activity may throw important light on all forms of cataract including the senile variety. The case for straightforward inheritance may be strengthened, or it may diminish and even disappear in the light of new knowledge in endocrinology and biochemistry.

Summary

An extensive new pedigree of familial cataract with Mendelian dominant method of inheritance is presented and the biomicroscopic appearance of the cataract is described. The possibility of other causal factors is discussed with the help of a review of the relevant literature, and a small series of blood analyses is presented.

Acknowledgments. I am indebted to the Wolverhampton Eye Infirmary for facilities to carry out the first year of the investigation and to Mr. H. B. Salt, M.Sc., of the Pathological Service, Worcester Royal Infirmary, for the blood analyses, by kind permission of Dr. McMenemey.

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A CASE OF RECURRENT APHTHOUS UVEITIS WITH ASSOCIATED ULCUS VULVAE ACUTUM (LIPSCHÜTZ)*

BY

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ISOLATED cases of this rare and interesting disease have been described under a variety of names from time to time. Franceschetti has traced one description as far back as 1772; occasional cases were reported towards the end of the nineteenth century; but it is only within the past fifteen years or so that it has been definitely recognised as a distinct clinical entity. Typically, the symptoms fall into three main groups: (1) periodically recurring uveitis; (2) skin eruptions of various types, *e.g.*, papular or papulopustular, *i.e.*, a papule resembling that of secondary syphilis with a pustular centre; recurring erythema nodosum—tender, rose-pink nodules in the subcutaneous tissue, some of which may be haemorrhagic; and transient subcutaneous nodules not involving the skin and associated with fever; (3) aphthous ulcers in the mouth and on the genitals, genital herpes, and, more rarely, the *ulcus vulvae acutum* of Lipschütz. In the male these aphthae develop on the glans, prepuce and scrotum, as well as in the mouth.

The disease affects young persons of both sexes, usually in the decade 20 to 30, and evolves slowly, by means of relapses, over a number of years. The three groups of symptoms constituting the syndrome are not necessarily present at the same time, and, as a rule, the eye symptoms appear to develop rather late. Once attacked, however, the eyes steadily deteriorate and progress, with remissions and relapses, towards blindness. One peculiarity of the disease as it affects the eyes, which is illustrated in the following description, is an alternating incidence of relapse from one eye to the other, suggesting an allergic mechanism.

Report of a case

G. M., female, aged 22 years, married, attended my clinic at the Central Middlesex County Hospital on December 5, 1944, complaining of pain and blurred vision in the left eye of three days' duration. Three teeth, which were said to have been septic, had been extracted five weeks previously. The patient neither smokes nor drinks, has always had good health, but said that she had lost weight recently. Her weight was then 7 st., 6 lbs. During

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her husband's absence in the Army she had been employed as a clerk in a Government department. She is an only child. Her parents are alive and healthy, except that her mother is said to have a weak heart. There is no personal or family history of aphthae. R.V.=6/9; L.V.=6/36 partly. The condition of the left eye was a mild acute iritis; no posterior synechiae, but the pupil dilated unevenly under atropine; there was colloid in the A.C., but no K.P. The blood W.R. was negative. A few days later the left macula was oedematous and there appeared a small patch of cloudy exudate at the periphery below. There were now large particles and much colloid in the A.C. and two or three spots of fine K.P.

By February 6, 1945, the media were much clearer. On February 20, however, the left vitreous was more hazy. There was no fresh K.P. but a circum-corneal flush was present for the first time and a new area of oedema just above the macula, with one small round haemorrhage. Mantoux reaction one inch diameter. The vitreous gradually cleared, but was again turbid on March 27. A report from the Ear, Nose and Throat Department said that there was no nasal obstruction or discharge; some chronic inflammation of both tonsils; X-rays of sinuses clear.

During the next month, apart from the appearance of a pigmented spot in the left macula, the eye continued to improve until April 17, when Cloquet's canal became hazy. The fundal periphery, however, remained clear. The blood sedimentation rate was: 1st hour /14 mm. (normal 5-8); 2nd hour /31 mm. (normal 8-14). She was examined by Dr. Joules and the chest X-rayed. A few adhesions were reported at both right and left bases and a calcified gland at the left hilum, but there was no evidence of pulmonary tuberculosis clinically or by X-ray. All the other systems were normal.

Early in July the right eye became inflamed for the first time. R.V. had fallen to 6/24. The vitreous was dusty, with cells in the A.C., and a small area of diffuse choroiditis near the macula, which was not visible with red-free (u.v.) light. The vitreous haze already noted in the left eye was still there, also the spot of pigment at the macula. A fortnight later the right macular lesion was smaller, but there was an increased shimmering reflex in this area. Somewhat later, on July 31, the haze in the left vitreous greatly increased. The right vitreous began to improve, but the enhanced macular reflex remained. The left vitreous became gradually more turbid. Several lesions could be seen in both fundi, but there was no more K.P.

At this stage the patient was very ill and felt giddy. She complained of night sweats, and the chest and abdomen were X-rayed,

with negative results. Cerebro-spinal fluid W.R. negative. Blood: Hb. 65 per cent.; W.B.C. 13,100 per c.c.; polymorphs 55 per cent., lymphocytes 36 per cent.; monocytes 5 per cent.; eosinophils 3 per cent.; basophils 1 per cent. Urine, acid; sp.gr. 1022; nothing abnormal found. Two teeth were extracted and this was followed by a rise in temperature, 101°2F. During the early part of September the evening T° regularly rose to 100°, and during the latter half of the same month to 103°F. She was admitted to hospital on August 23 and treated with sulphonamides. A total of 40 grms. sulphamezathine and 35 grms. of sulphathiazole was administered without any effect.

On October 16 the left eye suffered another acute relapse. Microscopic examination of the cerebro-spinal fluid revealed a few leucocytes and occasional hyaline casts. B.S.R. 12 mm./1st hour. A fortnight later the media of the right eye, which had been steadily clearing, suddenly became dull. The right eye, however, after this temporary lapse, again began to clear, but a new lesion (choroidal—not visible with red-free light) appeared in the macular region of the left fundus. During November both eyes became noticeably clearer, until on December 11 the patient reported that the right eye had been inflamed and " black " for a week. This proved the most severe of the recurrences in the right eye. There were two spots of fresh K.P. and the media were very turbid. The disc was obscured by exudate and vitreous haze. A large grey area, apparently of exudate, was present in the region of the macula. Some new, minute spots were seen in the left macula. B.S.R. 8mm./1st hour. The media of the right eye soon began to clear, but early in January, 1946, the patient again developed a temperature and complained of aching of the left upper jaw. Five teeth were extracted, happily without any local reaction or rise of temperature.

Some time towards the end of November or beginning of December, 1945, apparently about the time of the last severe relapse in the right eye, the patient had a rigor, which, she said, lasted on and off for three days, and was sufficiently severe to cause chattering of the teeth. Rigors recurred at intervals until April, 1946, altogether three or four. During these the patient remained at home and was not seen by me or by any of the physicians. Towards the end of January, 1946, she was confined to bed at home on account of pain in the left side of the chest. This was diagnosed as influenza. When she reported at my clinic on January 29 she was re-examined by a physician who found no evidence of pleurisy. Her temperature during this period was said to have risen to 102°F. This illness ended with a recurrence, accompanied by circum-corneal injection, in the left eye. B.S.R.

25 mm./1st hour. When I saw her on February 26 the media of both eyes, particularly the right, were clearing. R.V.<6/60; L.V.=6/60.

On this date (February 26, 1946) the patient reported for the first time a small vulval sore, situated on the left labium, which

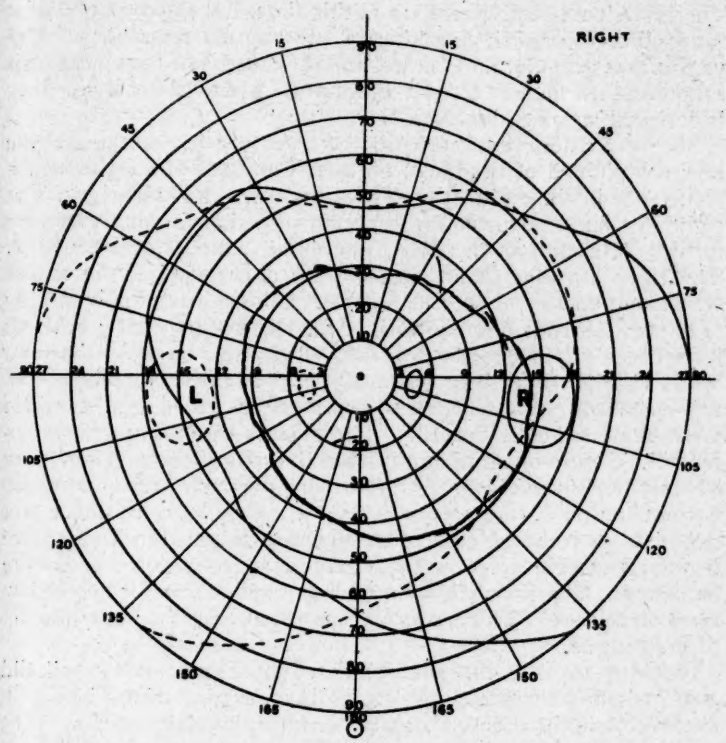

FIG. 1.

This chart, although taken in January, 1947, is representative of a series taken of the right eye during 1946. The object was a 3mm. white disk. It represents the peripheral limits of the field only, as it was found impossible to plot the irregular distribution of patches and gradients of vision and blindness which constitute the actual field.

was painful for five days but was now healed. At the same time there were small ulcers in the mouth. There were three or four of these, one on the left edge of the tongue and the others inside the cheek and inside the lower lip. The ulcers were roughly oval in shape, clean, shallow, and with sharply defined edges, approximately 2-5 mm. in length. A fortnight later the patient had a

severe uterine haemorrhage with clots and was pale but not collapsed. She was readmitted to hospital under a gynaecologist.

On examination no pelvic inflammation was found. By this time the patient's husband had been demobilised, but the patient denied the possibility of pregnancy and miscarriage. Subsequent events, however, suggest that this was the explanation. There was no vulval ulceration at this time, but the gynaecologist reported a " peculiar condition of the vaginal mucosa." The vagina was granular and tender, and the mucosa of the vault hard, rough and irregular. B.S.R. 15 mm./1st hour. After a few days in hospital she was discharged.

She was readmitted on April 3 for further investigation. She now complained of tiredness, night sweats, loss of weight, and a recurrence in the right eye. There had been a recent rigor. The right eye had one posterior synechia and the vitreous was very turbid. Throughout her stay the evening temperature was 99°·2. The right tonsillar gland was slightly enlarged, but the tonsils were small and were reported normal. Clinical examination of the chest and X-ray examination negative. Hb. 100 per cent.; W.B.C. 9,800; reds 4,250,000; platelets 425,000 approx; bleeding-time two min.; clotting-time three min., 20 sec. A catheter specimen of urine showed occasional leucocytes and reds; no organisms in a Gram film, and culture sterile. Throat swab—no haemolytic streptococci. Serum failed to agglutinate Brucella abortus. There were 31 units antistreptolysin " O " present per c.c., which is within normal limits. Mantoux 1 :10,000+ +. Capillary resistance test negative. A complete clinical examination was made with negative results, but five teeth were regarded as suspicious and were extracted. The socket of one of these was septic for some time after extraction. There was one small ulcer present on the tip of the tongue.

On May 6, 1946, the left eye flared up again. The pupil did not dilate and there was yellowish discolouration of the iris, with much colloid in the A.C. and corneal epithelial oedema. The tension of the left eye was soft. Owing to the corneal oedema and turbidity of the vitreous, the pupillary red reflex was poor, but a yellowish grey mass could be detected in the upper nasal region of the posterior vitreous. The right eye had not changed; there were still massive vitreous opacities and also the lesion at the macula already noted. For some weeks both eyes remained more or less in this condition, except that the red reflex improved somewhat in the left eye, although the mass in the vitreous was still present.

The patient was admitted to the Western Ophthalmic Hospital on July 12. The Mantoux reaction was over three inches in dia-

meter on the third day, and it was decided to give non-specific protein therapy. Two intra-muscular injections of milk (10 c.c. each) were followed by four intravenous TAB injections, the initial dose being 20 million B. Typhosus, 10 million B. Paratyph. A, and 10 million B. Para B. The right eye cleared very considerably and even the left eye began to show a brighter red reflex surrounding the exudation in the vitreous. This seemed to me the first response to treatment so far attained. Further, the fairly regular alternation of individual relapses, first in one eye, followed by an improvement, next in the other, had suggested to me for some time that there must be an allergic mechanism present, *i.e.*, an attack in one eye sensitising the uvea of the other and inducing a recurrence, which in turn became the excitor, and so on, in cyclic fashion. As I still suspected an underlying tuberculous infection I decided to attempt desensitisation by means of tuberculin injections. Accordingly, the patient was given weekly subcutaneous injections of old tuberculin, beginning with 1/1,000,000 mgrms., and very slightly and gradually increasing the dose. The improvement continued in both eyes. The right vitreous became very clear, apart from one or two moderately large opacities, and even the left seemed to be clearing satisfactorily, although the pupil remained irregular, the tension somewhat soft, and the A.C. shallow. The patient's general health had improved. The B.P. registered 120/60. About this time the patient announced that she was pregnant and this was confirmed by Mr. MacVine.

On October 16 the patient reported that she had noticed vulval ulceration for the past two months, and Mr. MacVine reported a small, rather painful ulcer situated on the left labium, for which he prescribed a 2 per cent. gentian violet paint. Within a month two ulcers developed at the same site and a contact ulcer, opposite the first, on the right side. The contact ulcer, however, rapidly healed and has not recurred. The original ulcer of the left pair grew rapidly, and was indurated, with raised edges, and a base composed of a thin layer of firm, slightly nodular, pale granulations, covered by a thin, light-coloured film. Mr. MacVine performed a biopsy, removing a wedge through the base of the ulcer, and inserting four catgut sutures. Dr. W. Pagel reported on the section as follows: " the ulcer goes fairly deep into the submucosa, its floor being formed almost completely by polymorphs. There is no clue as to the aetiology in the section examined. In the periphery of the ulcer the cells are chiefly lymphocytic: there are also some eosinophils and plasma cells. We are preparing a Ziehl-Nielsen section but will not report unless acid-fast elements are found." B.P. 136/70; B.S.R. 16 mm./1st

hour. Penicillin was administered locally and systemically, a total of 735,000 units, with no response. Later, the vulval ulcer was treated with a concentrated solution of urea. At the same time small ulcers, some with a sloughy base, appeared on the tongue and lips, especially on the right edge of the tongue. Culture from these yielded a few colonies of streptococcus viridans.

Just before treatment with penicillin, a rhinological report described some thickening of the lining membrane of both antra, particularly the left, and pronounced the frontal and posterior sinuses, and the posterior ethmoidal cells as normal. The hands were again examined by X-rays for cystic changes and were found normal. Analysis of the cerebro-spinal fluid gave protein 20 mgrms. per cent.; no excess of globulin; chlorides 720 mgrms. per cent.; W.B.C. 1 per cent. c.mm.; R.B.C. 250 per c.mm. The pressure was normal, and the Lange curve 0012110000. The W.R. was negative, and the complement fixation test (blood-serum) for gonorrhoea also negative.

It had now become clear that the diagnosis of this condition was the syndrome which is, perhaps, best described by the term recurrent aphthous uveitis, and that the vulval ulcer was the *ulcus vulvae acutum* of Lipschütz. Both eyes were still fairly quiet, the vulval ulcer very painful, and the aphthae in the mouth troublesome. On December 7, however, the left eye suddenly flared up and became quite blind. A necrotic, apparently encapsuled exudation occupied the whole of the posterior vitreous, giving rise to the amaurotic cat's eye reflex of Beer, and pressed forwards the lens, which remained clear, so that the A.C. was nearly obliterated and the pupil semi-dilated and fixed. A large emergent conjunctival vessel developed on the temporal side in the position of the ciliary body. The right fundus, on examination with red-free (u.v.) light, showed pale spots and streaks in the macular area, evidence of early micro-cystic degeneration of the retina. The optic disc looked very yellow—the patient is a blonde. There was a moderate amount of aqueous flare in the right A.C. and a large amount in the left. The larger of the vulval ulcers showed signs of healing, forming a waist across its centre. A further recurrence in the right eye was noted on February 24 and the media again became very turbid for some days. There were no ulcers in the mouth—the last was on February 18. Just before this last attack in the right eye the vision with correction (0.5 cyl. axis 150°)=6/36.

At first the foetal presentation was a breech, but towards the end of pregnancy it corrected itself. Mr. MacVine performed a Caesarian operation on March 6 and extracted a female child, weighing just over 5 lbs. but otherwise normal. During the night

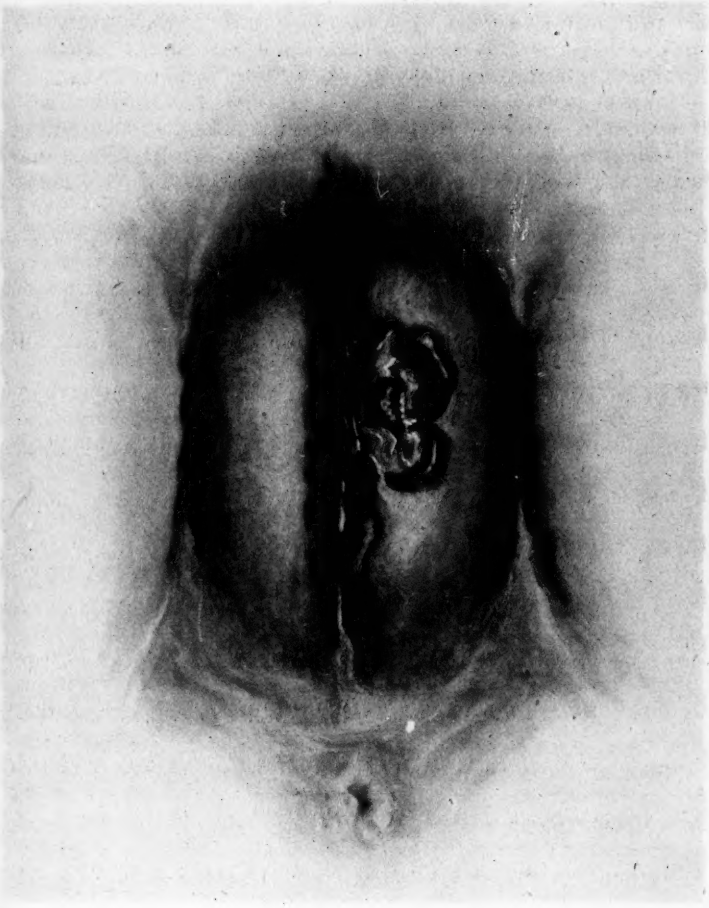

FIG. 2.

Ulcus vulvae acutum of Lipschütz. When the drawing was made the ulcer had begun to heal. Its maximum dimensions were 23mm. by 20mm. In the figure the left thigh is flexed and abducted in order to expose the ulcer fully.

of March 9 the patient had a severe rigor, accompanied by a temperature of 105°·2 F., and two more next day. Afterwards convalescence proceeded normally and the patient was discharged from hospital on March 26. A very mild and transient relapse occurred in the right eye shortly after the last rigor, and on March 19 the left eye became painful, interfering with sleep for about three days. On March 18 both vulval ulcers had completely healed with a normal and perfectly healthy scar. The patient felt and appeared to be remarkably well. A curious and perhaps significant point in connection with the vulval ulcers is that from first to

FIG. 3.

Microscopic section of ulcus vulvae acutum. Low power. Material from biopsy. Cell infiltration mainly lymphocytic but also with a good many plasma cells. Vessels typical of granulation tissue.

last, covering eight or nine months, there was no enlargement of the inguinal glands. Moreover, the second group of symptoms characteristic of this syndrome, *viz.*, skin lesions and eruptions, has been entirely absent. The patient has a fine, healthy skin, above the average, and at no time has there been a trace of papules, spots or other lesion.

Comments

I have given the history of this case in detail and at length, perhaps in too much detail and at too great length, but in describing an obscure and apparently rare disease—one which, so far as I know, has not been described before in British ophthalmological literature—with an unknown aetiology, it is impossible to foresee which of its manifestations will prove to be relevant in the light

FIG. 4.

The same as Fig. 3. Margin of the ulcer showing the epithelialized edge.

of fuller knowledge. During the long course of the disease my own attitude to its probable aetiology passed through three stages: (1) the focal sepsis phase; (2) the tuberculous phase; (3) the viral phase. The initial attack of iritis, which ushered in the disease, appeared then to be the ordinary iritis or iridocyclitis that is not uncommon in young adults. A history of tooth extraction a short time previously seemed to confirm this assumption, and further investigations were planned on this hypothesis, which was not shaken by finding a rather high blood sedimentation rate. Before

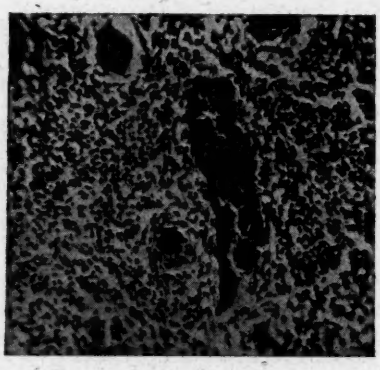

FIG. 5.

The same as Fig. 3. High power.

long, however, the loss of weight, night sweats and pyrexia, associated with a pronounced Mantoux reaction and high B.S.R., especially after the second eye had become involved, led me to suspect, even after clinical examination and X-rays of the chest had proved negative, an occult tuberculous infection. As the disease progressed, the apparent alternation of attacks from one eye to the other, lent colour to this suspicion, as it is generally agreed that sensitisation of tissues by tuberculous toxins explains some of its phenomena. The following table shows the dates of incidence from the beginning up to the time of writing:

TABLE I

1944	1945						1946				1947		
L. 2/12	20/2	—	31/7	16/10	—	—	29/1	—	7/5	7/12	—	—	19/3
R.	—	5/7	—	—	30/10	11/12	—	3/4	—	—	24/2	10/3	—

Altogether there have been fourteen attacks, eight in the left eye and six in the right. The series does not show exact alternation, but it is probably sufficiently close to conform with the theory of sensitisation. It may be remarked too that certain observers, *e.g.*, Stahli (1922), Urbanek (1934), and Meller (1934), who have reported cases of this disease, have advocated a tuberculous aetiology.

For over a year there were no aphthae in the mouth, nor as far as I knew, on the genitals. When buccal aphthae did appear, not sufficient attention was paid to them as the physician who was then in charge regarded them as dyspeptic ulcers. It must be remembered, also, that the teeth gave trouble on several occasions and some were extracted. This was a mistake of which I was not cognisant at the time, but, fortunately, there is no evidence that it provoked a focal reaction. The buccal ulcers, however, were at first more or less unconsciously associated with the condition of the teeth.

A remarkable feature of the relapses was their sudden onset and relatively short duration. Without any warning, as it were, an eye became affected acutely overnight, reaching its maximum intensity all at once, and then declining in a day or two. This also must be regarded as compatible with an allergic reaction, and I think it must be accepted that allergy does play a part in the symptomatology of this disease, although, as Franceschetti remarks, of a secondary character. Hypopyon, a characteristic feature of early descriptions, apparently of a transient character,

appearing and reappearing many times in the course of a single day, was never present in this case.

It was not until vulval ulcers appeared, some eighteen months after the initial attack in the left eye—apparently one or perhaps two vulval sores developed six months earlier, but were not reported by the patient until later—that the true nature of the condition became evident. The striking and very intractable ulcer, which developed on the labium majus, and proved absolutely resistant to every form of local treatment applied, was then recognised as identical with that described by Lipschütz in 1913 under the name *ulcus vulvae acutum*. Complete indifference to treatment, indeed, is the predominant feature of this disease. At one time non-specific protein therapy seemed to produce a favourable result. The patient herself was convinced of this, but the explanation of the apparent improvement then observed may quite well be, and probably is, that on this occasion the treatment coincided by chance with the beginning of a period of remission.

While Weekers and Reginster, in their second paper (1938), were the first to stress the independent nature of this disease, the credit for its complete elucidation belongs, in the first place, to Touraine (1941) and to some extent to Dascalopoulos (1932)—*l'uvéite récidivante aphteuse*—and, secondly, to Hulusi Bêhçet (1937) and to Franceschetti and Valerio (1939), who first advocated the theory of a viral aetiology. In support of this theory Franceschetti, Valerio and Babel (1946) state that recurrent aphthous uveitis has some resemblance to periodic ophthalmia in horses, which, however, has not been certainly proved to be a viral infection, and also to bovine aphthous fever, which is caused by a known virus transmissible to man.

It is not improbable that the pathogenic agents known as filtrable vira play a more important part in the causation of intra-ocular infections, *e.g.*, sympathetic ophthalmitis, also a disease of the young, associated with injury and resistant to treatment, than is commonly supposed. Vira vary in size; the largest are almost as big as the smallest bacteria; the smallest approach the size of large molecules. According to one school of thought the virus is a living organism; according to another it is an enzyme. Vira, however, exhibit the phenomenon of variation, which is a characteristic of living matter but not of enzymes. Essentially they are cell rather than body infections, as they appear to lack the enzymes necessary for independent growth. This peculiarity may explain their immunity from the effect of drugs. In other words, they are intra-cellular parasites, and in many diseases "inclusion bodies" are seen in the infected cells, *e.g.*, the

Guarneri bodies of smallpox; Negri bodies of rabies; Prowazek-Halberstaedter inclusions of trachoma, etc. They attack the cells of all three germinal layers, with the exception of muscle cells, and tend to infect young, healthy cells and young, healthy individuals rather than old, unhealthy ones. Rivers has explained that the part played by injury in causing viral lesions is due to the presence of young cells participating in the process of repair. For the same reason, embryonic cells are the best nutrient medium for their cultivation. The viral diseases constitute a large group of diverse conditions, such as measles, poliomyelitis, yellow fever, coryza, Rous' fowl sarcoma, etc. As a rule, one attack confers a life-long immunity, but influenza, the common cold, and herpes simplex are exceptions. They do not respond to sulphonamides, penicillin, or to any other known agent. Professor Bêhçet and his colleague Professor Braun, of the University of Istanbul, claim to have demonstrated inclusion bodies, or rather, the " elementary bodies," of which the cell inclusions are composed, in smears from the mouth and genital lesions of two cases. The smears were stained by the Giemsa and Herzeberg methods and formations resembling the elementary bodies of viral disease were found with each recurrence. Three patients, who also had aphthous ulcers of the mouth or genitals, were used as controls, and smears examined. Bêhçet and Braun, however, failed to find these bodies in the material from the controls. When stained with Giemsa, the bodies are approximately the same size as Guarneri bodies, *i.e.*, extremely small and round, reddish purple in colour, partly intra-cellular and partly extra-cellular. The same bodies were found after staining with Victoria-blue by the Herzeberg method.

These findings were observed on three different occasions and experiments were devised to determine whether they were artefacts or not. For example, smears dried in air and treated with alcohol-ether in order to extract any lipoids present, then stained with Giemsa and Herzeberg, showed abundance of these bodies. Also when treated with 3 per cent. acetic acid solution for fifteen minutes and stained, the picture was the same. After treatment with 3 per cent. sodium hydroxide solution for fifteen minutes, however, these bodies were no longer found. Smears taken from the vulval ulcer in my case by Dr. J. D. A. Gray and examined by Dr. F. O. MacCallum of the Wellcome Bureau of Scientific Research, proved negative. It should be noted, however, that when these smears were taken the ulcer was healing rapidly and for the present the negative result must be regarded as inconclusive.

Finally, attention should be drawn to one or two unusual features in my case. First, the fact of pregnancy. It is difficult even now to judge whether pregnancy and the metabolic changes

associated with it affected the course of the disease one way or the other. It is true that about the sixth month of pregnancy the left eye suffered its most severe attack and became blind, and that for the greater part of the period the vulval ulcer, not only showed no signs of healing, but became gradually worse, yet the general impression in my mind was that on the whole the effect was favourable. The ulcer began to heal during the last month or two of gestation and healed very rapidly after delivery. It may be recalled that there was a probable earlier miscarriage. Aphthae appeared from time to time during pregnancy. It is interesting to note here an observation by a colleague, *viz.*, that his wife who is subject to buccal aphthae, without other symptoms, during her pregnancies has always been free. The pregnancy of my patient was attended throughout by no untoward symptoms or discomfort.

Secondly, at one stage, lasting about five months, there occurred three or four severe and inexplicable rigors and again three days after delivery three rigors, apparently unconnected with the Caesarian section, for there was no evidence of uterine infection, followed one another within a period of about twelve hours. Otherwise, convalescence was uneventful.

Attention has already been called to the entire absence of skin symptoms; to the absence of leucocytosis; and to the absence of response to penicillin, sulphonamides, and other forms of treatment. Taken as a whole, the evidence, some positive and some negative, seems to point to infection by a virus as the most probable explanation of the nature of this disease.

Summary

The history of a case of recurrent aphthous uveitis is described over a period of twenty-eight months.

There were no dermatological symptoms but the *ulcus vulvae acutum* of Lipschütz was present.

Pregnancy occurred and went on to term.

Rigors occurred on several occasions.

Resistance to treatment is emphasised.

The hypothesis of a viral infection is discussed.

I have pleasure in expressing my thanks to Dr. Joules, the Medical Director, and to other members of the staff of the Central Middlesex County Hospital, especially Mr. MacVine, Dr. W. Pagel and Dr. J. D. A. Gray, for their invaluable help and co-operation.

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FURTHER EXPERIENCE WITH AMNIOTIC MEMBRANE GRAFTS IN CAUSTIC BURNS OF THE EYE*

BY

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IN an earlier communication (Sorsby and Symons, 1946) attention was drawn to the value of amniotic membrane grafts in the treatment of caustic burns of the eye. In a series of 30 cases (the burn being produced by lime in 22 cases; by hydrochloric acid, sodium hydroxide, and ammonia in 2 cases each; and by liquid sulphur dioxide and lead in 1 case each) no corneal or conjunctiva sequelae were noted in 27 cases. In 28 eyes in which vision was known 26 showed vision of 6/9-6/5. A method of applying the graft was described and it was indicated that early grafting gives the best results. Generally speaking the eyes were found to be very nearly white in 3 to 5 days after grafting and they were usually normal by the end of a week.

1.—A further series of 28 cases

Table I sets out the distribution and the clinical details in a further series of 28 cases. These fall into three distinct groups:

* Received for publication, May 5, 1947.

1. LIME BURNS (21 cases).

(a) Severity.—The severity of the burns can be judged by the following data:

Conjunctiva:

Both upper and lower fornices burnt	3
Lower fornix only	16
Inner half of conjunctiva	1
Lower fornix and outer half of conjunctiva ...	1

Cornea:

Clear	1
Whole of cornea involved	2
Three quarters of cornea involved	3
Half of cornea involved	6
Lesser areas of cornea involved	9

(b) *Time of Grafting.*—Grafts were invariably applied on the same day as the patient was seen. In 9 cases this corresponded to the day of the burn; in 10 cases it was a day after the injury was sustained. In one case the patient was not seen until 2 days after the injury, and in one more until after 4 days.

(c) *Duration of Treatment.*—Eight of these 21 cases were treated as out-patients. The time taken for the eye to become normal is shown in the following summary table:

Eye normal in 2 to 5 days	10 cases
,, ,, ,, 6 to 8 ,, ...	7 ,,
,, ,, ,, 9 to 12 ,, ...	0 ,,
,, ,, ,, 3 to 18 ,, ...	3 ,,
Unrecorded	1 case

(d) *End result.* (1) *Cornea.*—In all but two cases the cornea was clear on discharge. In the two cases in which there was a corneal opacity a pre-existing corneal scar was present in one (No. 18), whilst delay in treatment appears to have been the responsible factor in the second one (No. 1), the one case in which the graft was carried out 4 days after the burn.

(2) *Vision.*—Apart from the one feature with a pre-existing corneal scar in whom vision was 6/24, vision was good, or could

be presumed good in the remaining cases. It was 6/6 to 6/5 in 12, 6/9 in 5 cases, and it was not recorded in 3 more, 2 of whom were illiterate children.

(e) Complications and sequelae.—Apart from the corneal opacity in case 1 already noted, a tendency to symblepharon was noted in 2 cases (Nos. 13 and 16). In each case this tendency was suppressed by grafting; in case No. 13 the one graft used was adequate, whilst in No. 16 a further graft had to be inserted after 48 hours. In two more cases a second graft was found necessary: in case 9, treated as an out-patient, the graft was found under the bandage when the eye was dressed after 48 hours; in case 19 there was the unique experience of more staining of the conjunctiva being present on removing the bandage after 48 hours; in both these cases there was an uneventful recovery after a second graft.

2. SODIUM HYDROXIDE (5 cases).

Three of the 5 burns produced by caustic soda can be dismissed briefly. The lower fornix was involved in 2 cases and the inner half of the conjunctiva in the third; the cornea was involved to the extent of ¼, ½ and ¾ respectively. The grafts were all applied on the day of the burn. Two cases became normal in three days and one required 9 days. The cornea was clear in all cases and vision was 6/5 in one case and 6/9 with correction in the two others.

Considerably more severe injuries were noted in the two eyes of a man, aged 64 years. (Nos. 25 and 26.) When seen three hours after an explosion, in which his eyes were burned, both cornea were pearly opaque. Amniotic grafting was carried out on the same day, and subsequently twice more at intervals of 4 days, and finally once more 6 days later. A hypopyon developed in the right eye 20 days after the burn, when paracentesis and anterior chamber lavage were carried out. After prolonged treatment vision of the right eye was reduced to perception of light; a dead-white opacity of the cornea was present. In the left eye the lower two-thirds of the cornea is intensely opaque and vision is counting fingers at one metre.

3. FIREWORKS (2 cases).

Of the two injuries produced by fireworks one was a superficial conjunctival burn on the left side with three-quarters of the cornea staining. There was no conjunctival burn on the right side, but one-eighth of the cornea showed staining. Amniotic membrane grafting was carried out on the left eye on the day of injury with an excellent result.

TABLE I

Clinical details on caustic burns treated by amniotic graft.

No.	Age	Extent of cornea involved	Conjunctival fornices involved	No. of day grafted after burn	No. of days to become normal after grafting	END RESULT		Remarks
						State of cornea	Vision (with correction where applicable)	
					1. LIME BURNS			
1	28	½	Lower	4	17	Scar	6/9	First seen 4 days after injury.
2	30	⅓	Lower	0	5	Clear	6/6	—
3	19	¼	Lower	1	2	Clear	6/5	Out-patient.
4	30	¾	Upper and lower	1	4	Clear	6/6	—
5	16	½	Lower	0	6	Clear	6/6	—
6	42	¾	Lower	1	8	Clear	6/6	—
7	23	½	Lower	0	3	Clear	6/5	Out-patient.
8	51	¾	Lower	0	5	Clear	6/6	—
9	32	½	Lower	1	5	Clear	Not recorded	Two grafts.
10	17	¼	Lower	1	5	Clear	6/9	Out-patient.

10	17	¼	Lower	1	5	Clear	recorded 6/9	Out-patient.

TABLE I—*continued.*

Clinical details on caustic burns treated by amniotic graft.

						END RESULT		
No.	Age	Extent of cornea involved	Conjunctival fornices involved	No. of day grafted after burn	No. of days to become normal after grafting	State of cornea	Vision (with correction where applicable)	Remarks
11	33	½	Lower	0	6	Clear	6/9	Out-patient.
12	52	Complete	Upper and lower	0	Not recorded	Clear	6/9	Out-patient.
13	13	None	Lower	1	5	Clear	6/6	Tendency to symblepharon suppressed by grafting.
14	33	¼	None	1	3	Clear	6/5	Inner half of bulbar conjunctiva involved.
15	6	¼	Lower	1	3	Clear	Not recorded	Outer half of bulbar conjunctiva also involved.
16	6	¼	Lower	0	7	Clear	Not recorded	Tendency to symblepharon suppressed by a second graft.
17	30	⅓	Lower	1	6	Clear	6/6	Out-patient.
18	64	⅙	Lower	2	13	Old scar	6/24	Out-patient. Old corneal nebula.
19	31	¼	Lower	0	8	Clear	6/6	Out-patient. Two grafts. More staining after first graft.
20	53	½	Lower	1	7	Clear	6/6	—

TABLE I—*continued.*

Clinical details on caustic burns treated by amniotic graft.

No.	Age	Extent of cornea involved	Conjunctival fornices involved	No. of day grafted after burn	No. of days to become normal after grafting	END RESULT: State of cornea	END RESULT: Vision (with correction where applicable)	Remarks
21	31	Complete	Upper and lower	0	18	Clear	6/9	Central corneal opacity. Intense chemosis at first grafting. Lime found present on first dressing 2 days later; grafted again.
				2. SODIUM HYDROXIDE BURNS				
22	47	½	Lower	0	9	Clear	6/9	—
23	33	¼	Lower	0	3	Clear	6/9	Out-patient.
24	41	¾	None	0	3	Clear	6/5	Inner half of bulbar conjunctiva involved.
25	64	Complete	Upper and lower	0	—	Opaque	P.L.	Extensive opacification of cornea at injury.
26						Opaque	C.F.	
					3. FIREWORKS			
27	11	Complete	Upper and lower	1	49	Opaque	C.F.	Extensive burn of cornea and sclera. Immediate opacification of cornea.
28	17	¾	Lower	0	7	Clear	6/9	—

The second case was a severe burn involving the conjunctiva, episclera, sclera, and cornea of the right eye giving staining of the whole of the cornea with a total opacity. Though the final result was counting fingers in that eye, the clinical course during treatment and convalescence was not unsatisfactory. There was no marked tendency towards symblepharon, nor was there any worsening of the initial condition of the eye.

2.—Discussion

1. *Significance of early grafting.*—In the previous communication the difficulty in obtaining experimental control of the findings recorded was indicated. Nor could adequate clinical control be employed. Some approach to clinical control is, however, available by comparing the results obtained in the first series with those recorded in the present series. The 22 lime burns in the first series were submitted to grafting by amniotic membrane at variable times after the injury. At the beginning early grafting was exceptional, and it was only towards the end of the investigation that grafting on the day of the injury was undertaken. Classifying the data previously recorded the following summary table emerges.

Number of day after burn when grafting carried out.	Duration in days of in-patient treatment for each case respectively
0	5, 2, 4, and no in-patient treatment in two cases
1	5
2	10, 8, 7, 12, 10, 6, 2, 5
3	17, 15, 11, 27
4	9
5	18
6	21
10	17

These results in themselves bear out the suggestion made in the earlier communication that early treatment is advisable. This suggestion is strengthened by the data in the following

summary table on the present series extracted from Table I to show the time taken for the eye to recover its normal state in relation to the time when the graft was applied after the burn:

Number of day after burn when grafting carried out.	Number of days for eye to become normal in each case respectively
0	5, 6, 3, 5, 6, ?, 7, 8, 18*
1	2, 4, 8, 5, 5, 5, 3, 3, 6, 7
2	13
4	17

* Case No. 21. Lime found in eye at first dressing of amniotic graft.

The fact that in both series late grafting involved prolonged treatment is indicative of the value of the procedure.

2. *Mode of Action.*—The experimental results previously reported suggested that human amniotic membrane was species specific in its action, and possibly tissue specific too, in so far as it reacted well with conjunctiva but not with skin. There is no support for species specificity or tissue specificity from experiments with tissue culture (Sorsby and Ungar, 1947). It would appear that amniotic membrane does not stimulate growth, but that it acts as a sort of internal splint for the proliferating tissue.

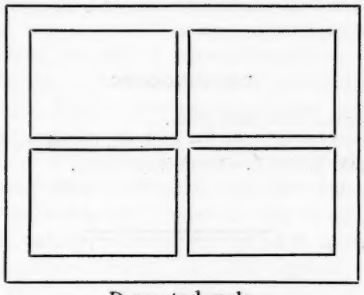
3. *Mode of use.*—The method previously described has stood the test of further use. Occasionally the procedure of inserting amniotic membrane has been modified in particular instances to cover defects in the conjunctiva not readily served by an implant into the fornix. In such cases partial covering of the cornea has sometimes to be used. In most cases such partial covering does not appear to act deleteriously in contrast to total covering of the cornea; moreover, the membrane generally tends to retract leaving the cornea fully exposed.

4. *Limitations of amniotic membrane grafting.*—Cases Nos. 25, 26 and 27, which illustrate immediate and severe damage to the cornea, show the limitations of amniotic membrane grafting. It is necessary to distinguish clearly between immediate (and presumably irreversible) damage to the cornea and secondary involvement of the cornea from a persistent raw conjunctiva.

Amniotic membrane grafting will have no effect on the first; it is invaluable as a preventative of the second type of corneal damage. A crude parallel is the cornea in ophthalmia neonatorum. The sulphonamides and penicillin have little effect on the damaged cornea; they are invaluable in preventing corneal ulceration and infection.

5. *Method of preparing amniotic membrane grafts.*—We are indebted to Mr. H. P. Morley of the Ligature Department, The London Hospital, for the following note on the mode of preparing amniotic membrane grafts of human origin. In his view the method of preparation is sufficiently simple to be possible in any well equipped pathological laboratory or dispensary.

(1) The placenta should be placed in normal saline immediately after delivery, the following process begun within twenty-four hours.
(2) Carefully separate the amnion from the chorion, commencing from the edge of the fringe up to the cord.
(3) Wash the amnion thoroughly in running water, then place in a saturated solution of common salt for twelve hours.
(4) Remove the amnion and immerse in a fresh saturated solution of common salt for twelve hours.
(5) Extend the amnion on black-backed plate glass and remove mucus and surface fat with gauze swabs.
(6) Wash thoroughly until the membrane is free from salt and immerse in distilled water from some ten to thirty minutes, frequently agitating the solution.
(7) Immerse the amnion in a 1 in 150 solution of potassium hydroxide (caustic potash) frequently agitating for at least one hour. No membrane being constant, the time factor is governed by the appearance of the membrane and experience.
(8) Extend the membrane on the plate glass and wash with distilled water, removing soaps formed.
(9) Pour hydroxide solution on the membrane and remove all remaining fat by manipulative massage with gauze swab. If necessary, further hydroxide baths and manipulative massage should be applied.
(10) Wash the membrane free of alkali.
(11) Test for fat by re-saponification.
(12) Immerse the membrane for twelve hours in distilled water, then stretch on wood frames measuring—outside measurements—6½ in. × 8½ in., divided into four sections. The 6½ in. × 8½ in. frames can be made of smooth, polished wood measuring ½ in. × ½ in. and the wood dividing the frame into four, ½ in. wide by ½ in. deep.



Drawn to ¼ scale.

The membrane will adhere naturally to the frames.

(13) Allow to dry at ordinary room temperature, then remove from the frame and trim.

(14) Sterilisation is effected by dry heat at 150°C. for one hour in open-mouthed cellophane envelopes.

Summary

(1) A further series of 28 cases of caustic burns of the eye treated by grafting with human amniotic membrane is recorded.

(2) There were 21 cases of lime burns. In 10 of these the eye was normal within 2 to 5 days, and in 7 more within 6 to 8 days. In all cases but one the cornea was clear at the end of treatment. Vision of 6/9-6/5 was recorded in 17 cases, and could be presumed in three more. In two cases a tendency to symblepharon was controlled by grafting.

(3) There were 5 cases of burn due to sodium hydroxide, and 2 to fireworks. In 3 of these 7 eyes there was severe immediate corneal damage. This was uninfluenced by amniotic membrane grafting, though grafting affected favourably the course of these eyes.

(4) Taking the previous and the present series together it is shown that remarkably rapid response may be expected when grafting is applied on the same day, or within 24 hours after the injury; subsequently, recovery is slower.

(5) The mode of action of amniotic membrane grafting is not clear. It would appear that the amniotic membrane does not stimulate growth, but acts as a sort of internal splint for the proliferating tissue.

(6) The method of preparing amniotic membrane, as practised by Mr. H. P. Morley, at the London Hospital, is described.

We are indebted to Sister Hollands and Sister Thorogood of the Ophthalmic Unit at Lambeth L.C.C. Hospital for their help with the treatment of these cases.

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VISUAL DIAGNOSIS OF EYE DISEASES BY MEANS OF INFRA-RED RADIATION[1]

BY

A. VAŠKO *and* M. PELEŠKA

PRAGUE

LATELY we have been experimenting with image converters[2] which are sensitive to infra-red radiation in an attempt to aid the diagnosis of certain diseases of the eye. We are studying normal as well as pathological tissues of the anterior and posterior parts of the eye and also investigating the possibility of seeing through opacities of all the component parts of the eye (cornea, aqueous humour, lens and vitreous humour).

For this purpose we measured the spectral transmission characteristics of these media. Fig. 1 shows the spectral distribution of transparence of a very opaque cornea and lens, the former

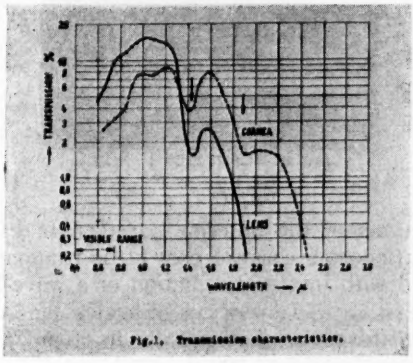

FIG. 1.

having been removed during a transplantation. The characteristic was measured on a double rocksalt monochromater shortly after the operation. Fig. 2 shows the same cornea before the operation in visible light and Fig. 3 in infra-red light. Various degrees of opacity of lenses and corneae resulted in non-uniform spectral transmission characteristics, but the transparence always increases as we move from the visible to the near infra-red region and measurements would indicate up to now a maximum of the transparence of opaque lenses in the region of wave-length $\lambda = 10{,}000 - 12{,}000$ Å. The arrows in the diagram indicate absorption bands of water.

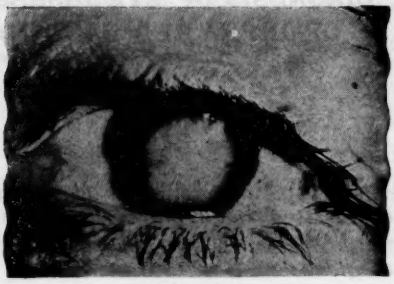

FIG. 2.

FIG. 3.

For observations of the anterior parts of an eye through an opaque cornea (leucoma corneae post ulcus traumaticum) the eye was illuminated with infra-red radiation of a wave-length exceeding 8,500 Å and by means of an objective lens an image was projected on to the photo-electric cathode of an image converter with a long-wave limit of 14,000 Å. This image converter transforms the infra-red image of the observed eye on the cathode into a visible image on a fluorescent screen. This method of direct observation of the movable eye presents a number of advantages against photography on infra-red sensitive plates. We can simply focus by visual observation of the image on the fluorescent screen and photograph the image on the screen when the position of the eye is most suitable. Localisation in depth inside the eye can be estimated by employing parallactic movement of the eye. Also we can use infra-red radiation of longer wavelength than can be used in direct photography, because the long exposures involved in photography on infra-red sensitive plates render this process impracticable for the living eye; therefore the infra-red

photographs which are known from literature all use merely the nearest parts of the infra-red spectrum.[3 7]

The infra-red image converter which we used had also been adapted for use in conjunction with the customary apparatus for ophthalmological diagnosis (Nordensen slit-lamp).

Observation in infra-red light proved to be especially useful as a complement to the conventional methods of ophthalmological diagnosis before transplantations of the cornea and similar, nowadays frequently performed operations.

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"THE RETICULO-ENDOTHELIAL SYSTEM OF THE CORNEA"*†

BY

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POLAND

THE results of investigations on colloidopectic properties of corneal cells are divergent. Whereas some writers as Blotevogel, 1924, Gasteiger, 1934, and others state that cells of the corneal stroma undoubtedly possess a colloidopectic ability, others as Loehlein, 1925, Jirman, 1936, did not succeed in confirming the observations of the above authors. Judging the question as one of far reaching theoretical and practical importance experiments were started on the permeability of cornea for some colloidal substances. Appropriate dye solutions and silver compounds were applied to the conjunctival sac. I attempted also to find those cells of the cornea which have the strongest properties of storing electronegative colloids, *i.e.*, which cells of the cornea

* Received for publication, April 25, 1947.

† From the Eye Clinic of M. Curie-Skodowska University. Director: Professor Dr. I. Abramowicz.

may show properties of elements of the reticulo-endothelial system.

Thirty white rabbits were used for the experiment. In 20 rabbits solutions of appropriate acid dyes, 1 per cent. trypan blue, izamin blue and carmin after Kiyon and alkaline dyes, 1 per cent. neutral red were dropped into the conjunctival sac during a period of 50 days.

The remaining 10 rabbits were treated with silver compounds 1 per cent. sol. arg. nitr., 5 per cent. protargol solution and 25 per cent. argyrol solution. Rabbits which had received acid and alkaline dyes revealed macroscopically a slightly discernible coloration. In microscopic preparations, however, no traces of dye deposits were found. In unstained sections 100-200μ thick a rather uniform tint was stated which was undoubtedly the result of infiltration of all corneal layers with the applied dye which penetrates through the cornea into the anterior chamber similarly as through a half permeable membrane. Cells of the corneal stroma do not show colloidopectic abilities when colloidal solutions of acid and alkaline dyes are applied to the conjunctival sac.

That some corneal cells may reveal colloidopectic properties is proved by results of further experiments in which silver compounds were used. In contradiction to hitherto prevailing opinions it appeared that silver deposits in corneal argyrosis may

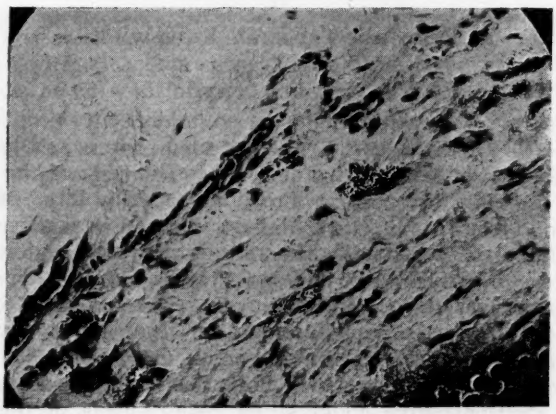

FIG. 1.

Corneal stroma. Stroma cells with numerous silver granules visible. Magnified ×550.

occur almost exclusively in Descemet's membrane and that silver in form of deposits may occur in all corneal layers as well, except the epithelium. Silver deposits are partly located intracellularly within stroma cells, *i.e.*, in sessile corneal cells partly outside the cells within structures of reticulin fibres in Bowman's and Descemet's membrane. Largest silver deposits occur within Descemet's membrane. These abundant deposits of silver granules within the elastic membrane are undoubtedly a consequence of some affinity of silver compounds to the so-called reticulin structures. Considerably smaller silver deposits in Bowman's membrane are most probably due to a partial resorbtion of silver granules by stroma cells. These transport the silver by means of plasmatic ways to the second reticulin membrane which constitutes a sort of a last barrier on which granules of silver compounds are held. This series of experiments proves that corneal stroma cells show distinct storing properties for silver, colloidopectic, and may be in some cases transformed into typical poliblasts, see micro-photograph.

We may therefore look upon corneal stroma cells as wandering cells in state of rest (Maksimow), which means that they correspond to prohistiocytes. Considering the above mentioned properties of stroma cells we may range them among elements which do not differ functionally from the fundamental components of the reticulo-endothelial system.

Summary

Colloidopectic abilities of the corneal stroma cells have been stated by means of a one per cent. Arg. Nitr. solution applied to the conjunctival sac of rabbits during a longer period of time. Stroma cells exhibit distinct properties in storing silver and are transformed in some cases into typical poliblasts. The opinion that stroma cells correspond to wandering cells in a state of rest seems to be justified. Corneal stroma cells may thus be ranged among elements of the reticulo-endothelial system.

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OPHTHALMIC CALCULATIONS BY THE "DAM" METHOD

BY

JOSEPH I. PASCAL

NEW YORK

IF one should ask for example the average ophthalmologist what is the power of a contact lens having given radii of say 8·5 and 8 mm. and made of a given substance, say, n=1·488 the chances are he would be stumped even though he had once learned the method of procedure. Most likely the same reaction would be encountered if he were asked to calculate the power of the cornea or of an ordinary ophthalmic lens.

There is a method for calculating surface power and lens power which is really very simple and once learned can never be forgotten. I have called it the " Dam " method, and I don't mean " damn." It is based on the formulation of a unit of curvature comparable to the unit of power which is the dioptre.

Everybody knows that a 1·00 dioptre lens has a focal length or briefly a focus of one metre or 100 cm., or 1,000 mm., or approximately 40 inches. It is sufficiently accurate to take one metre as equal to 40 inches. The relation between power in dioptres and focal length, or briefly focus, is expressed by these " key " numbers. To get power from focus or focus from power we divide into one of these key numbers. For example, what is the focus of a 4·00 D. lens ? Divide 4 into 100, gives 25 cm.; divide 4 into 1,000, gives 250 mm., divide 4 into 40 gives 10 inches. Or conversely, what is the power of a lens having a focus of 20 cm.? Divide 20 into 100, gives us 5·00 dioptres. If the focal length is given in inches, say, the focus is 20 inches, divide 20 into 40 which gives 2·00 D. and so on.

Exactly the same relation that exists between power (in dioptres) and focus in metres, centimetres, millimetres, or inches, exists between curvature and radius in the same units. The radius just like the focal length may be expressed in metres, centimetres, millimetres, or inches. The curvature is expressed in a unit of curvature called a metrec (term derived from metre-curve). The relation between curvature (in metrecs) and radius is expressed by exactly the same key numbers as used in the relation between power (in dioptres) and focal length. Thus what is the curvature of a surface having a radius of 20 cm.? Divide 20 into 100 and we get 5 metrecs. Or what is the curvature of a surface having a radius of 8 mm.? Divide 8 into 1,000 and we get 125 metrecs. Or what is the curvature of a surface having a radius of 10 inches ? Divide 10 into 40 and we get 4 metrecs.

The " Dam " formula is the general formula for surface power and is best written as D=aM. " D " stands for dioptres of power, " a " stands for the difference between the second index (that is the index of the medium where the light goes to) and the first index, *i.e.*, the index of the medium where the light comes from. It can be written as " a " equals N_2-N_1. It may be memorised that " a " stands for the amount the second index is *a*bove the first index; M stands for the curvature in metrecs. For example, what is the power of the anterior surface of the cornea having a radius of 8 mm. and an index of 1·376? The radius and index must be given or assumed. Using our " Dam " formula we get " a "=1·376−1=0·376, and $D=0{\cdot}376\times\frac{1000}{8}=\frac{376}{8}$ =47 D. Or what is the power of the *posterior* surface of the cornea given a radius of 6·8 mm. and the index of the aqueous as 1·336? Again D=aM, in which " a " is the difference between the second index, the aqueous humour on the other side of the surface, and the first index, which is that of the cornea itself (the posterior surface of the cornea separates the cornea from the aqueous humour). Here " a " equals 1·336−1·376=−0·040.

$$D=-0{\cdot}040 \text{ times } \frac{1000}{6{\cdot}8}=\frac{-40}{6{\cdot}8}=-5{\cdot}88 \text{ D.}$$

The " D=aM " formula gives the power of a surface and combination of surfaces in terms of reduced power. This is the power used by Gullstrand and other writers on physiological optics. For a surface bounded by air, or for a lens as ordinarily used, *i.e.*, in air, the reduced power is the same as the actual power and we need not go any further into this.

In contact lens calculations since the radii are always given in millimetres the radius has to be divided into 1,000. If one takes " a " to three decimal places and then moves the decimal 3 places, which multiplies it by 1,000, one has only to divide this by the radius to get the power. Thus if a contact lens made of n=1·488 has radii of 8·7 and 8·5 mm. the surface powers will be $\frac{488}{8{\cdot}7}=+56{\cdot}09$ for the anterior convex surface and $\frac{488}{-8{\cdot}5}=-57{\cdot}41$ for the posterior concave surface. If the lens is infinitely thin the total power is the sum of the surface powers, in this case −1·32 D. In contact lenses the effect of thickness cannot be ignored and one must use a formula involving thickness.

This, however, is a secondary consideration and has to be used only after the surface powers are obtained. The " Dam "

method after being used for a couple of examples gives a simple and easily retained method for all lens problems. Say what is the power of a thin ophthalmic biconvex lens of radii 25 cm. and 40 cm., made of glass index being 1·52? Using D=aM for first surface, we get D=·52 times $\frac{100}{25} = \frac{52}{25}$ =2·08 D. For posterior surface figuring that surface is convex to the outside, and the first index is air, we get D=·52× $\frac{100}{40} = \frac{52}{40}$ = 1·30 D. Total power is +3·38 D.

The formula for the total power of a lens of any kind where the thickness has to be taken into account is not difficult to remember if one but thinks about it for a few minutes. The total or combined power is the sum of the surface powers with a modifying term. The formula consists of three terms, two of which are the surface powers and the third is the modifying term. Thus $Dp = D_1 + D_2 - D_1D_2e$. Dp stands for combined principal power, D_1 is the power of the first surface, D_2 is the power of the second surface. The sum of these two gives the basic power of the whole lens. The " modifying " third term is the product of these two powers, times the equivalent thickness " e." The equivalent thickness " e " is the actual thickness expressed in metres and divided by the index of refraction. Thus if the thickness is 3 mm. and the index is 1·50, e = $\frac{0{\cdot}003}{1{\cdot}5}$, or if the thickness is ·5 mm. and the index 1·488, " e " equals $\frac{0{\cdot}0005}{1{\cdot}488}$.

In short, the sum of the surface powers, minus their product times " e " gives the total power. The formula is best remembered when read just as written, " Dp equals D one plus D two minus D one D two e." In the contact lens example given above assuming the thickness to be 1 mm. the modifying third term is 56·09 times −57·41 × $\frac{·001}{1{\cdot}488}$. This works out to +2·16. Adding 2·16 to the sum of the surface power −1·32 gives us a true principal power of +0·84 D.

The effect of thickness in this instance is very marked. In most contact lenses it is very much less marked. In ophthalmic lenses in general the effect of the thickness is negligible. So that if one forgets everything except the " Dam " method he will still be able to calculate all surface power, all thin lens power, and at least approximately nearly all " thick " lens problems.

A NOTE ON THE POSITION OF THE EYE IN A THIRD NERVE PALSY*

BY

EUGENE WOLFF *and* Surgeon Commander HEFFERNAN

LONDON

IT is generally stated that in a complete third nerve palsy the eye looks out and somewhat down or simply down and out due to overaction of the external rectus and superior oblique.

We would submit that this statement requires some qualification. The eye is certainly abducted but as in this position the superior oblique cannot depress the eye or only very slightly, the amount of depression is minimal or nil as in the following case :—

On November 18, 1946, G.W., aged 21 years, was involved in a collision while motor cycling.

FIG. 1.

* Received for publication, February 27, 1947.

He was taken to hospital and did not recover consciousness for five days.

When he came to, he noticed that he could not open his left eye. There was no other paralysis but he was told that he had had a temporary left sided hemiplegia for the first three days. He noticed that when he lifted the left upper lid with his hand he saw double, The diagnosis was fracture of base of skull, nasal bones and left jaw with left-sided third nerve paralysis.

When seen on January 20, 1947, we found as follows :—

Left eye closed, but he could lift the lid very slightly with the aid of the frontalis. Vision 6/6 in each eye.

Left pupil larger than right and fixed to light and convergence.

With the right eye looking straight forward and the left lid lifted manually (Fig. 1) the left eye was seen to be directed *directly outwards. The corneal reflexes were on the same horizontal level.*

On directing his right vision to the extreme right the left eye came to the mid-line. No other movement of the left eye was possible except in-torsion on asking the patient to look down. Both fundi were normal,

WAREN TAY-SACHS DISEASE IN A CHINESE INFANT*

BY

GOPAL HARIDAS

(FROM THE CIVIL GENERAL HOSPITAL, SINGAPORE)

A SURVEY of all the available literature on Tay-Sachs disease fails to reveal evidence of the record of a case previously from Malaya. I am thus submitting a full report of a case, which was admitted to the General Hospital, Singapore—the first case of its kind occurring in Malaya in a non-Hebrew infant—together with a brief review of the clinical types, history and symptomatology which I believe will not prove repetitious in spite of the extensive and controversial literature that has already been written on this subject.

Tay-Sachs disease is the infantile form of amaurotic family idiocy. Amaurotic family idiocy is a type of progressive cerebral degeneration which comes under the large group of the cerebro-retinal syndromes of the heredo-degenerative type. The following most modern classification of the clinical types of the cerebro-retinal syndromes of the heredo-degenerative type is given in the British Encyclopaedia of Medical Practice, Volume III :—

* Received for publication, March 29, 1947.

1. Tay-Sachs Disease (Infantile amaurotic idiocy).
2. Batten-Mayou Disease (Juvenile amaurotic idiocy).
3. Late Infantile Cerebro-Retinal Degeneration.
4. Late Juvenile Cerebro-Retinal Degeneration.
5. Anomalous Types.
   (*a*) Tay-Sachs Disease without typical macular appearance.
   (*b*) Batten-Mayou Disease with the cherry-red spots.
   (*c*) Amaurotic idiocy without amaurosis.

Histological summary

In 1881, Waren Tay, an English ophthalmic surgeon, examined a 12-month-old male baby who was brought to him because he was unable to hold up his head and to move his limbs. At first, he made a provisional diagnosis of defective cerebral development and later examined the fundi. The examination revealed in the yellow spot in each eye in Tay's words " a conspicuous tolerably defined, large white patch more or less circular in outline and showing in its centre a brownish-red circular spot, contrasting strongly with the white patch surrounding it." Tay pointed out that this red spot was not due to haemorrhage or pigmentation but seemed to be due to thinning of the retina, thus forming a small hole or gap in the white patch which exposed the vascular choroid. When he examined the infant 4½ months later, the macular appearances were unchanged but the optic discs which were first normal had become atrophic. The child had also become helpless and died when he was 20 months old. Subsequently three more male cases were observed by him in the same family and they died before the age of two years.

In 1887, Bernard Sachs of New York published a full report of a case of a peculiar form of idiocy associated with blindness. Later, a much younger sister of this patient also became similarly affected. The fresh brain at post-mortem was found to be unusually hard and the knife actually grated on removing a small section of the cortex. A histological examination of the cut cortex showed the protoplasm of the pyramidal cells to be in various stages of degeneration.

In 1894, Kingdon pointed out that Tay's description of the ocular changes and Sachs' description of the nervous manifestations both formed the important features of one and the same disease and in the same year Carter drew attention to the fact that the disease was confined to the Jews. In 1896, Sachs named the condition amaurotic family idiocy, and the name Tay-Sachs was suggested by Higier in 1901.

In 1909, Schaffer studied the histo-pathology of this disease

in great detail. He characterised the minute pathology as an unusual swelling of the cell protoplasm and the dendrites. The interfibrillar material is first attacked and the cell degeneration follows. There results degeneration of every cell of the central grey matter of the brain, spinal cord and spinal ganglia and of the ganglion cells of the retina. There is attenuation of the nerve-fibre layer. The nerve cells later become filled with a granular pre-lipoid material. Schaffer concludes by expressing the opinion that children with amaurotic family idiocy possess " an abnormally exhaustive nerve-cell protoplasm, which, becoming paralysed with the strain of earliest functions, soon degenerates."

As the result of a few most important observations and elaborate microscopical examinations made by Poynton, Parsons and Gordon Holmes (1906), and by Mott (1907), the following conclusions were arrived at by them :—

(1) That amaurotic family idiocy is a primary disease of the nervous elements and that the proliferation of neuroglia is secondary to this.

(2) That it is a primary cell disease because the nerve-cells are relatively more affected than the fibres.

(3) That the primary change is in the interfibrillary part.

They also concluded that :—

(a) The disease was not due to arrested development because the symptoms were not present from birth.

(b) The disease was not due to bacterial toxins.

(c) The disease was due to some inherent biochemical changes in the protoplasm of the cell which led to its degeneration.

Recently histo-pathologists have shown that this disease is one of a group of primary metabolic disorders which leads to an accumulation of lipoid substances, chiefly phosphatides in the nerve cells of the brain and ganglion cells of the retina.

Aetiology

Nothing definite is known of the cause of this affection. Syphilis, tuberculosis, alcoholism and neurotic taint play no part in its production. Infection has been excluded. It has been definitely established that it is a familial and racial disease. The occurrence of 111 cases in 69 families containing two or more members and the presence of consanguinity in the parents in more than 50 per cent. has brought out the recessive character of the condition. There is undoubtedly a greater incidence of this disease among the children of the Jewish race, especially the Jews of Poland and Russia, probably because consanguineous marriage is common

among this race. Slome (1933) has traced 18 undoubtedly non-Jewish cases among the 200 or so reported in the literature. The case I am about to describe is a Chinese Hylam infant. A few cases have also been noted among the natives of Japan. Kamel Yacoub (1938) has reported five cases of Tay-Sachs disease, all in one Egyptian family.

Symptomatology of a typical case

The "clinical picture" of Tay-Sachs disease is so characteristic that when once seen it will never be forgotten. The infant is normal when born and develops normally up to the third or sixth month. He then becomes listless and apathetic and no longer seems to take notice of objects. As the weakness increases he is unable to sit up. The neck muscles are weak and his head falls backwards or forwards if unsupported. There is a progressive diminution of vision and the eyes wander aimlessly about. He is susceptible to sounds. There is optic atrophy and the cherry-red spot surrounded by a lighter halo at the macula. The limbs now become rigid, the arms are extended and rotated inwards at the shoulder, the legs are extended, adducted and crossed. The knee-jerks at this stage are usually exaggerated and the plantar reflex may give an extensor response. The cerebro-spinal fluid is normal. Wasting gradually becomes extreme. The hands become flexed and contracted. The feet become extended; the knee-jerks at this stage may be difficult to obtain. The infant lies in a semi-conscious condition, convulsions may occur and a squint may be present. If the infant is fed nasally he may be kept alive for some months in an emaciated condition. Death takes place from pneumonia or quite suddenly from cardiac failure.

Relation of Tay-Sachs disease to Niemann-Pick disease

The pathological changes in Tay-Sachs disease may serve as a basis of an opinion as to the relationship of this condition to Niemann-Pick disease. In 1928, R. Hamburger observed the occurrence of Tay-Sachs disease in a case of Niemann-Pick disease. In 1928, Bielschowsky made a histological study of a case and drew attention to this association of which a number of instances have been reported.

Niemann-Pick disease is a rare condition usually met with in Jewish children. Symptoms appear within a few weeks or months after birth. The spleen is considerably enlarged, the liver becomes enlarged and the infant becomes wasted and pale. Death occurs before the end of the second year.

Histological examination of the liver and spleen shows the presence of large "foam cells" distended with a yellowish lipoid.

Similar cells are also found in the bone-marrow, suprarenal cortex and lymphatic glands, while the ganglion cells of the nervous system may be stuffed with lipoid. It is said that this lipoid in the ganglion cells does not represent the product of degeneration of the nerve-cell, but a faulty general metabolism with fat deposited in the nervous tissues. There is a striking similarity in the chemical composition of the abnormal cell contents in the liver in Niemann-Pick disease and of the brain in Tay-Sachs disease. Pick, Spielmeyer, Bielschowsky and Kufs have expressed the belief that Tay-Sachs disease has as its basis the same metabolic disturbance. However, in Tay-Sachs disease there is localisation of the general disturbance to the central nervous system alone. But Baker and Platou as the result of pathological findings in a case of amaurotic family idiocy they studied, are not inclined to agree with above view that Tay-Sachs disease and Niemann-Pick disease are related. They are inclined to agree with Schaffer (1935) that Tay-Sachs disease is an endogenous condition involving ectodermal elements and is not related to Niemann-Pick disease. Schaffer says that in Tay-Sachs disease there results a primary degenerative condition with swelling of nerve cells which later become filled with a granular pre-lipoid material and that Niemann-Pick disease was the result of a metabolic disorder. Von Bogaert's observation of cases of Tay-Sachs and of Niemann-Pick diseases, in the same family, is strong evidence in favour of similar pathology in these two diseases.

In addition to the symptoms already described, a large proportion of cases show mental degeneration, spastic paralysis, fits and blindness, in short the symptoms of amaurotic family idiocy and further the pathology of the nervous system in both diseases is identical. It seems probable that Niemann-Pick disease and amaurotic family idiocy are merely different clinical expressions of one and the same condition.

Report of the Case

Register No.: 464/40. Hoo See Ngee, a male Chinese Hylam infant, aged 17½ months, was admitted to the Children's Ward, General Hospital, Singapore, on March 3, 1940, at 11.30 a.m. with a history of convulsions off and on for 4 months.

History: The infant was delivered normally at full term. History regarding consanguinity was not obtained because the parents never came back to see the infant after it was admitted into hospital. The infant is the fourth child in the family. The eldest child, a male, is healthy and his intelligence is normal. The other two children are dead and the cause of their death is not known. The infant was brought to hospital because he had been having fits for the last four months. He had been breast-fed for

three months after birth and then he was condensed-milk-fed. Rice water was given for some time. The child had been able to move his limbs, cry and recognise objects and mother up to four months before admission—that is until he was 13½ months old. He has never been able to sit up by himself but he could be made to sit up when supported by pillows. The mother said that the infant became less and less active as days passed on and up till four months ago she did not notice anything abnormal except that he was unable to sit up.

Examination.—He weighed 16 lbs. 14 ozs. on admission. He appeared pale and wasted. The circumference of his head was 46 cms. His head was large. He lay in bed listless and apathetic and took no notice of his surroundings. He did not follow a bright light and he took no notice of the feeding bottle. His eyes when opened were noticed to wander aimlessly about. His eyebrows were devoid of hair. He made occasional involuntary sucking movements with his lips. His mouth was kept open with everted lips.

Objects placed in his hand were not grasped. He could not take feeds from the bottle because he would not suck at the bottle. He would not swallow milk and medicines given with the spoon but they would only accumulate in the mouth. They had to be administered by means of the nasal catheter.

He lay in whatever position he had been made to lie without making the slightest attempt to move his limbs or turn on either side. The muscles were flaccid and toneless and the limbs were slightly rigid. One could occasionally notice some movements of the fingers of the right hand but the limbs as a whole were paralysed. The deep reflexes were exaggerated and the plantar reflex was flexor.

If he was made to sit up his head fell forward. He was sensitive to noises.

An examination of the fundus with the ophthalmoscope conducted under chloroform anaesthesia by Dr. A. D. Williamson, the ophthalmic physician and surgeon, General Hospital, revealed marked pallor of both discs, a somewhat oval dark purplish-red spot at macular areas (about ¼ size of disc) on a milky bluish-white background about three times area of disc.

An X-Ray examination of the skull, conducted by Dr. J. W. Winchester, Radiologist, General Hospital, Singapore, showed that the fontanelles were widely opened and that there was no direct evidence of tumour.

Progress: The infant used to have frequent attacks of convulsions. His head gradually became bigger—size of head on

Marked pallor of both discs; a somewhat oval dark purplish-red spot at macular areas (about ¼ size of disc) on a milky bluish-white background about three times area of disc.

March 3 was 46 cm. and on May 23 was 49·8 cm.—indication of a rapidly increasing hydrocephalus; on May 23 anterior fontanelle measurements

were 7 cm. × 7 cm.
5 cm. 5 cm.

On March 12 he had an attack of acute naso-pharyngitis and on March 15 he had enteritis. He got cured of these two conditions. On March 21 he developed a cellulitis of the scalp which gradually became worse.

Muscular wasting progressed to an extreme degree. The limbs became more rigid and all the deep reflexes were exaggerated. The arms were extended and rotated inwards at the shoulder, the legs were extended, adducted and crossed. The hands became flexed and contracted and the feet extended. There were numerous pressure sores all over the body. He had been kept alive by nasal feeding. He developed pneumonia and died of it on June 8, 1940,

that is when he was 20 months and 20 days old. An autopsy was performed but the autopsy notes are not available.

Laboratory examinations.—Blood cholesterol 186 mgm. per cent.; Blood Kahn Test Neg. (000); cerebro-spinal fluid Wassermann reaction Neg.; Cerebro-spinal fluid clear, not under pressure, nothing else abnormal.

Treatment: Antuitrins 1 c.c. daily intramuscularly for 6 days; and large doses of potassium salts.

Comments

This case of the infantile form of cerebro-macular degeneration is worth recording because it is the first case of its kind from Malaya in a Chinese infant. In the absence of a complete history, it is difficult to exclude the familial nature of the illness. Two other children in the same family have died, but it is not known of what they died. Although the age at which the first signs and symptoms manifested themselves is not clear and definite, the clinical features and course of the disease should be characteristic enough to make a clinical diagnosis possible but this was offset by the fact that this was a non-Hebraic case which confused the racial incidence and also by the fact that no such case had been seen before in Malaya. He was delivered normally at full term. He was admitted to hospital for convulsions when he was 13½ months old. But before this the mother had noticed that he had not been able to sit up by himself and that he had become less and less active as days passed on. After admission, it was noticed that there was marked muscular weakness and wasting, signs of visual failure ending in blindness and marked lack of mental development. An examination of the fundus revealed signs pathognomonic of the infantile form of cerebro-macular degeneration, a somewhat oval dark purplish-red spot at macular area on a milky bluish-white background. An additional feature in this case is a rapidly increasing hydrocephalus. He stayed in hospital for three months and five days and died when he was twenty months and twenty days old of terminal lobar pneumonia.

Summary

1. A case of the infantile form of cerebro-macular degeneration, otherwise known as Waren Tay-Sachs disease or amaurotic family idiocy, in a Chinese Hylam male infant is reported, the first of its kind from Malaya.

2. Due to an incomplete history, no history of consanguinity or of familial incidence was obtained.

3. The child was twenty months twenty days old when he died.

4. Diagnosis was established from the progressive muscular weakness, lack of mental development and blindness with pathognomonic changes in the fundus.

In conclusion, I have to thank Dr. William Heng for assisting me with the notes of this case, Dr. Chew Poh Kuo for the original diagram of the appearance of the left fundus, and the Director of Medical Services, Singapore Union, for permission to publish this case.

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TECHNICAL IMPROVEMENTS IN THE DIATHERMY OPERATION FOR DETACHMENT OF THE RETINA

BY

PROF. A. KETTESY

DEBRECEN, HUNGARY

THE diathermy operation for retinal detachment, in spite of its great development, still has such technical difficulties, which may be eliminated by improvement of the instruments used in the principal phase of the operation, the coagulative closing of the hole by the pointed perforating electrode.

There have been two main forms of perforating electrodes in use up to date: the needle, as described and used by Weve, Vogt, Szily-Machemer and others, and the nail of Safar and Arruga.

The drawback of the needle is that it has to be pulled out after every puncture. There arise leaking channels through the sclerotic and choroid from the beginning, with more or less continuous loss of inter-retinal fluid. Their number increases during the operation necessarily, hence the eye softens and the scleral surface

cannot be kept dry. Both circumstances make the further punctures more and more difficult, till at last it is sometimes impossible to continue with diathermic perforations, and one is obliged to change to other means, *e.g.*, to the cautery-loop.

Attempts have been made to avoid these inconveniences by shortening the working length of the needle and by refining its point. The depth of the puncture should not exceed 1·0—1·5 mm., so as to reach the choroid, but not to pierce it. Yet, this form had its own disadvantages, too. Too small an area of coagulation behind the retina could not always be seen by the ophthalmoscope, hence the control became doubtful. Moreover the coagulative effect would not suffice sometimes to arouse the necessary adhesive inflammation.

The nails, inserted, remain in place during the main phase of the operation. The Safar nail is still the best, yet set into the sclerotic its head protrudes 2 mm. above the scleral surface. Whenever the eye is turned back into the primary position—necessary for ophthalmoscopic control—the head of the nail is caught in Tenon's capsule. In consequence of this the head is either laid flat down on the sclerotic or lifted out. In the first case the point of the nail is vacillating inside, a great danger to the retina and the vitreous. Laid flat down on the choroid its discovery with the ophthalmoscope is rendered difficult. On the other hand, if taken out or loosened, it brings about the inconveniences of the filtrating channel. Besides it has eventually to be sought in the tissues. (Therefore nails should be as a rule always counted.)

There have been still more drawbacks in Safar's set. His insulated forceps did not seize the nail firm enough. During the insertion it readily tilts over. The forceps is too clumsy, its faces are too large, and sometimes it is difficult to get deep enough with it into a narrow slit.

All disadvantages can be eliminated by giving a better shape to the nail and by constructing suitable forceps for it.

This new nail is made of highly polished stainless steel. It has a round flat head, not thicker than 0·3 mm. and with a diameter of 1·5 mm. The length varies from 2 to 6 mm. It begins cylindrically and passes conically into a sharp point (Fig. 1).

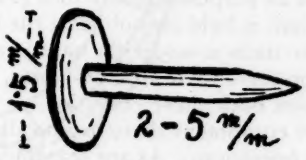

FIG. 1.

The new diathermic nail magnified.

The new insulated forceps has angular ends. On the inner side of each end a small groove runs all along the three margins (Fig. 2). The parallel grooves work together and are designed to catch the nail.

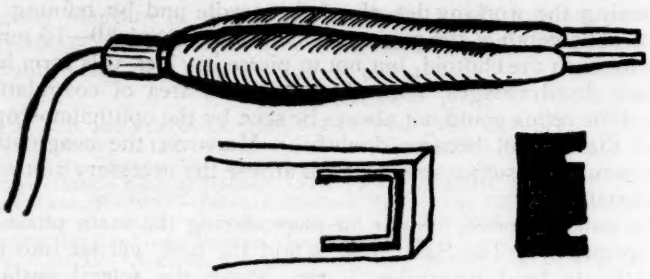

FIG. 2.

The isolation-forceps in full-shape, the grooved inner surface of the end and its cross-section.

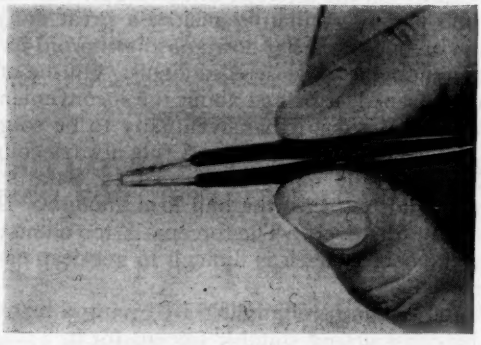

FIG. 3.

The nail seized with the isolation forceps perpendicularly.

The nail is easily seized by this forceps and directed as well in the axis of the forceps as perpendicularly to it (Figs. 3 and 4). The head of the grasped nail is held immobile in the forceps.

In order to keep the nails arranged at hand, and to protect them from being lost, we preserve them in cork-plates, taking them from there and putting them back there (Fig. 5).

Working with this equipment there are no disturbing moments in the course of the operation. As we use the nails not only for the closing of the hole, but at the same time for localisation, some

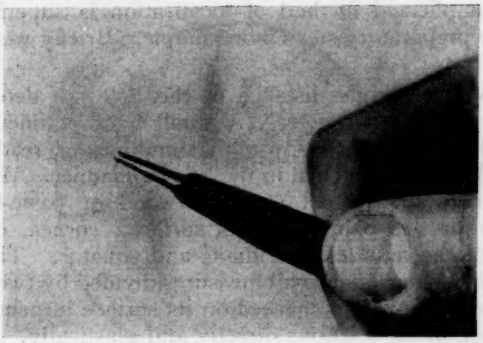

FIG. 4.
The nail seized vertically.

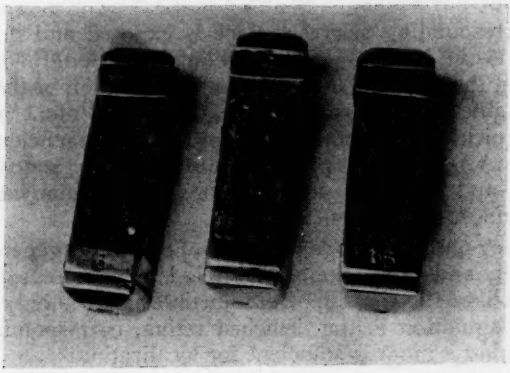

FIG. 5.
The nails arranged on cork plates.

FIG. 6.
Small perimeter.

separate complicated method of localisation is superfluous, thus making the preparatory steps more simple. Briefly we proceed as follows:—

Meridian and limbus distance of the hole are determined by estimation and the perimeter. A small hand-perimeter (*Fig. 6*) allows direct ophthalmoscopy with simultaneous reading of the degrees. A plan is sketched in the known manner. We found an agreeable help an eyeball made of wood or paste-board. All necessary data are painted on its surface: cornea, ora serrata, insertions of the muscles, meridians and equator. The diameter of the ball is 44 mm., hence all measures divided by two are giving natural size. The hole is marked on its surface in pencil, thus we see the topographic relations directly and plastically.

We do not instil cocaine, thus diminishing the danger of the corneal surface becoming hazy. A drop of pantocaine suffices to be able to make the local anaesthesia with subconjunctival injections of novocaine.

The corresponding scleral surface is exposed and dried. The first nail is set in following the plan. The length of the nail depends on the diastasis of the retina in the region of the hole, yet it is advisable to choose a long one, say 5 or 6 mm., in order to find it quickly by ophthalmoscopy. With sufficient current (20—30 mA.) the nail plunges into the eye almost without resistance and the head adheres closely and immovably to the scleral surface.

With the ophthalmoscope the point of the nail is generally found immediately and its relation to the hole is established. With luck the nail strikes the right spot. Sometimes we see only the white spot of coagulation in the detached retina, corresponding to the needle behind it; that is sufficient for localization.

If the first nail is in a wrong position we settle the place of the next nail by estimation, *e.g.*, 3 mm. backwards meridianally and 2 mm. temporal or nasal longitudinally. The nail put in, new ophthalmoscopic control follows: It is superfluous to say that all nails set in remain preliminarily in their place. If necessary, we put in a third and fourth nail, till we did hit the hole or its margin somewhere, thus being able to get an easy start to put in all the other nails around the hole. These nails are shorter, 2—3 mm.

An example of an operation is shown in Fig. 7.

There is a typical hole of horseshoe form, meridian 2.30 o'clock, 13 mm. from the limbus. The first needle of 5 mm. hits the middle of the hole. With the following needles of 3 mm. length we mark the main points of the planned lines of coagulation, checking their

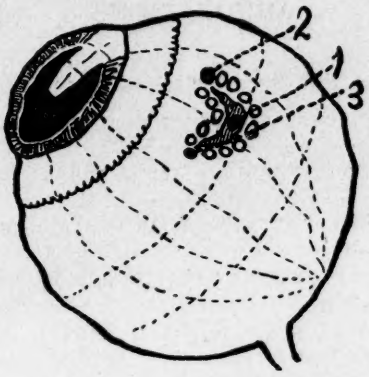

FIG. 7.

Plan and course of a detachment operation.

right position by ophthalmoscopic examination we fill up the lines with nails of 2 mm.

Having put in the last nail, an ophthalmoscopic control may follow, yet is not necessary.

The nails are quickly removed one after the other with the grooved forceps, and the operation field closed. (The nails are counted.)

There arises no harm from the nails in the wrong position or having perforated the retina. All such points give rise to small local inflammations, contributing to the adhesion of the re-attached retina.

It did not seem to be necessary to insulate the head of the nail or the upper part of its shaft, though it could be done. Bare nails compared with insulated needles did not show any difference in working.

For more than 3 years I have been using this set. Having gone through the whole evolution of the detachment-operation in the practice, I find it is superior to all other sets from the technical point of view. We make ophthalmoscopic examinations of the patient lying on the operating table and we start the operation only when we are certain of finding every detail of the fundus quickly and surely in this unaccustomed position. This and the technique together have contributed much to shorten the operation and to keep the cornea clear. The operation has become more exact, and this manifests itself in the increased percentage of the successful cases.

ANNOTATIONS

On the Decay in Proof Reading

It is odd that the more experience one has of reading proofs the worse one becomes at spotting typographical errors. We can only suppose that familiarity breeds contempt and that as one gets older one automatically pays less attention to what one reads. That we are not alone in this practice is evident in the perusal of an article in one of the daily newspapers of March 29, 1947, where the compositor has printed *habeus corpus* for *habeas corpus* and whoever read the proofs slipped up over the difference between "u" and "a." It is an easy mistake to make. But if one reads a book published in the early years of the last century one usually finds the proof reading to have been extremely good. Even with the old fashioned "long s," which is so closely similar to the letter "f," we did not notice a mistake in two volumes of about 500 pages each. Here, if anywhere, an error would be almost excusable. In the old days when the type was set by hand it should have been easy to pick out an "f" from the upper stratum of the case, and a "long s" from the lower stratum. And in using new type no error should be possible; but with old type which has been repeatedly in action it would be no wonder if, when the type was broken up, an occasional "long s" got into the "f" compartment and *vice versa*.

Maybe the having had to concentrate on type of this sort will have helped to improve our proof reading. It was quite curious to note how one would make mistakes over the "long s" and take the word "seed" for "feed." On rare occasions either would have made sense; but when we read what, at first sight, appeared to be "muffins" at the beginning of a line, we had mentally to scratch our head and look again. The word on closer inspection was "muslins." The paragraph dealt with the manufacture of commodities and however succulent a muffin of that era may have been it was obviously out of place in this connexion.

On Bindings

Our thoughts this month are devoted to bindings, by which we mean the covers of books not the binders that swathe the abdomen of the recently delivered female.

In olden times it was not unusual for the sheets to be sent out from the press bound in boards so that the purchaser could have the book bound in any style or material he liked. Plutocrats would choose a binding they liked and have the majority of the books in

their libraries bound alike. We have always thought that the results of this proceedure were monotonous and would prefer to have different bindings for different sizes of books at the least. Some books are of much greater value uncut and in their original boards than in the most expensive form of binding.

A great deal depends on whether the owner wants his books to be read or merely looked at; some bindings, like some persons, wear very much better than others. School prizes used often to be bound in "tree calf," a form of binding that looks nice and shiny when new, but does not wear well. You do not need to be an authority on pig skins or law calf to settle the material for binding. Indeed, nowadays the question is settled for you by the publisher.

Provided a text-book be not too large and weighty, cloth forms a good binding. If a stouter cover is needed, buckram is excellent. Vellum, or half vellum, in any colour you like, gives a very good appearance and wears well. As to colour, this of course is infinite. The publisher will provide any colour in reason. We even remember having seen a book on colour blindness the cover of which was half red and half green. There is something to be said for a standard colour for professional text-books. Fuchs' first English translation came out in black cloth; the edition we bought in 1907 was in green, then came the red colour which has persisted till today. Nettleship's little book was in green, Jessop's in blue, Henry Juler's in black and Sir George Berry's in green.

No one would wish to see the colour of a long set of volumes such as the *Transactions of the Ophthalmological Society* changed from its perennial green. Long may it continue! We always wondered who chose the colour of Vol. I. Was it Sir William Bowman, or the Council, the secretaries or the publishers? Search of the early minutes might answer the question, but like most of the utterances of Mr. Toots, it's of no consequence.

Much more important than the binding is the size and legibility of the print. It seems to us a poor economy to put a badly printed book into expensive covers for distribution as school prizes: but as these, we understand, are only to be looked at and never to be read it does not matter very much.

Their name liveth for evermore. *Ecclesiasticus, xliv,* 14

It is sometimes said that our profession is too fond of tacking the surname of anyone who makes a discovery on to it. The process can certainly be overdone, but there are some nominal appellations we could ill spare.

The circle of Willis, the Eustachian tube and the foramen of Monro are cases in point. And, may we, in parenthesis, remind

Southerners that the great Edinburgh anatomists did not spell their name phonetically?

Go where you will in medicine and its allied subjects you meet examples of what we are considering. The pharmacists have their Blaud's pill, Dover's powder and Easton's syrup among others. Our own specialty contains many examples and the names of Bowman, Meibomius, Argyll Robertson, Beer and von Graefe are always cropping up and their is hardly any telling where Hutchinson may not appear. We believe that Briggs was the first to describe the optic papilla, but if ever it had his name attached to it we have choked him off. Schlemm's canal is indispensable, and Herbert's pits save a deal of descriptive writing.

When all is said, we are no worse offenders than any other profession. A botanist who discovers a new flower, a sportsman who shoots a new antelope, an ornithologist who finds a rare bird, each is sure to have his name added to it as a memorial. And, for ourselves, we have always liked the principle. Hunter's canal must have been familiar to anatomists from very early times, but Hunter first made use of it to place a ligature on the femoral artery on the proximal side of a popliteal aneurysm and deserves an honourable tribute of this kind which will endure for all time. We see no reason why we should not continue to praise famous men in this way and the fathers that begat us.

BOOK NOTICE

The 1946 Year Book of the Eye, Ear, Nose and Throat. The Year Book Publishers Inc. Chicago, $3·75. (Obtainable from Messrs. H. K. Lewis & Co., 136, Gower Street, London).

This new edition of the well-established Year Book of the Eye, Ear, Nose and Throat deals with the literature from August, 1945 to July 1946, and follows the lines of its predecessors. The contribution to ophthalmology occupies about 300 pages and is compiled by Dr. Louis Bothman of Chicago. The review of the year's literature is not, of course, comprehensive, but the author is to be congratulated on the choice of papers referred to as well as the readable way in which the subject matter is presented. The volume will be found useful to ophthalmic surgeons, but so far as practice in this country is concerned, it would be more so if it were divided into two with a complete separation of oto-rhino-laryngology from ophthalmology.

CORRESPONDENCE

THE "NORMAL" IN THE SYNOPTOPHORE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—I am glad to see that Mr. Emsley, in his letter in your last issue, confirms the very points that I, in my letter in your February issue, wished to make.

If I had said "qualities (as opposed to quantities) of heterophoria" I should perhaps have been more clear. It is precisely because it is not different "qualities" of heterophoria that the two instruments test, that the failure to check the zero on the synoptophore assumes importance when comparing the instruments.

We should, I think, all agree that the quantity of heterophoria revealed by any test depends upon the method of dissociation used. It depends also upon many other factors, some of which I have tried to examine in an earlier paper (*Brit. Jl. Ophthal.*, p. 142, 1941) to which I would refer Mr. Emsley. My plea is for precision, where precision is possible, for an unchecked instrumental error obscures the very factors it is desired to measure.

Yours faithfully,

NIGEL CRIDLAND.

SOUTHSEA, HANTS.
May 22, 1947.

COLOUR VISION IN THE CONSULTING ROOM

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—In the article by Dr. Neubert on "Colour Vision in the Consulting Room," May, 1947, p. 275. There are several points which require further explanation. The gross incidence of colour defectives is given in 5·5 per cent. which is considerably below the most recent estimates of the condition for the male population (7-8 per cent.). This low incidence may be due to the method of testing, no clear description of which is given. It is important to remember with the Ishihara plates that the light source must be good daylight or the test is of no value. With the Giles Archer Aviation Colour Perception Unit it is difficult to standardise the test, and no mention is made of the Test-Retest

reliability of this instrument on the same subject. This is a fundamental point where comparisons of different apparatus are to be made. In the later stages of the paper a testing lantern with three lights is quoted, but no mention is made of the constitution of this lantern. The only item of its construction which is given is that the source of light was regulated by a rheostat. Now such a method of control prevents adequate control of the light intensity and is also responsible for variations in the colour temperature of the source. Once again there is no reference to the test—retest reliability of the second type of lantern, and without that no useful comparison can be made. Finally, no attempt has been made to determine whether the second lantern is testing exactly the same thing as the first—colour contrast plays a part in the second lantern and not in the first, hence the two lanterns are not testing exactly the same thing.

In the absence of such information relating to the tests reported in this article, the author's conclusions about the relative sensitivity of the single light and the multilight lanterns are rendered invalid.

Yours faithfully,

JOHN GRIEVE.

MEDICAL SCHOOL, DUNDEE.

May 19, 1947.

NOTES

Excerpta Medica WE have received an announcement of this new publication together with the first issue of Section 13 (Dermatology and Venereology). The headquarters are in Holland and the Editor in chief is Dr. W. M. Woerdeman. The intention is to furnish a complete survey of all medical literature in the form of short abstracts. These abstracts are prepared by a staff of 3,000 specialists supervised by 400 editors. No details as to subscription rates appear in the announcement but these can be obtained from 111, Kalverstraat, Amsterdam, C.

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Appointments AT the last Council meeting of the Faculty of Ophthalmologists, Mr. J. H. Doggart was nominated the representative on the Editorial Board of the British Journal of Ophthalmology in place of Mr. Goulden.

H. B. Stallard, A. G. Cross and A. G. Leigh have been appointed Assistant Surgeons to the Moorfields, Westminster and Central Ophthalmic Hospital.

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The Illuminating Engineering Society. Annual General Meeting.

AT the Annual General Meeting of the Illuminating Engineering Society, on May 13, the President, Mr. J. S. Dow, presented the Report of the Council for the year ending December 31, 1946. Though recovery to normal conditions has not been as rapid as might be desired the Society has continued to make good progress both as regards activities and membership. For the first time the Council were able to report that membership has passed the 2,000 mark.

In the year under review a Centre has been opened holding new meetings alternately at Gloucester and Cheltenham.

The outstanding event of the year was the Society's first Convention, held in London, which proved a great success. The Society is now making arrangements for a Summer Meeting to be held at Harrogate from May 26 to 29, 1948, when the programme will include papers and social events. It is hoped that this initial meeting will be followed by other periodical gatherings in other areas.

The names of the following Officers for the next session were announced:—

President:—Dr. J. W. T. Walsh; *Vice-Presidents*:—Mr. J. M. Waldram, Mr. J. S. Preston and Dr. E. C. Walton; *Hon. Treasurer*:—Mr. J. C. Holmes; *Hon. Secretary*:—Mr. H. C. Weston; *Hon. Editor*:—Dr. S. English.

Dr. Walsh, the President-elect, is Chairman of the National Illumination Committee of Gt. Britain and is a Principal Scientific Officer in the Light Division of the National Physical Laboratory. He is well known to lighting engineers throughout the world as the author of several works on Illumination and Photometry which, though published over 20 years ago, are still regarded as standard works on the subject.

His election sets a new precedent, for this is the first time that any member of the Society has twice been elected to the Presidential Chair.

Finally the President announced the result of a ballot of members that had been taken on the proposal to form a Register of Lighting Engineers. Under the scheme the names of those corporate members who fulfil certain conditions may be included in a Register and are entitled to the exclusive use of the description "Registered Lighting Engineer (I.E.S.)." This proposal had been approved by a large majority.

On conclusion of formal business the President introduced Dr. N. A. Halbertsma, President of the International Commission on Illumination, who delivered an address entitled "International Relations in Illuminating Engineering." Dr. Halbertsma outlined the history and development of the Commission and explained the method of working of the Executive Committee and the National Committees of the various member countries. He also gave a number of amusing anecdotes to illustrate the difficulties met on account of the different languages spoken by delegates representing many countries at meetings of the Commission. Finally,

Dr. Halbertsma mentioned problems still before the Commission and mentioned that it was hoped that more countries will become members of the Commission in the future.

The next meeting of the International Commission on Illumination, the first since the outbreak of war in 1939, has been arranged to take place in Paris in September, 1948.

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Oxford Ophthalmological Congress

THE XXIIIrd Annual Meeting of the Oxford Ophthalmological Congress will be held in Oxford, on July 3, 4, and 5, 1947. Mr. Williamson-Noble will be invested as Master on July 3, in the Department of Human Anatomy and will welcome the visitors. After the annual general meeting a discussion on "the contracted socket" will be opened by Professor Pomfret Kilner of Oxford and Mr. H. B. Stallard, of London. Mr. Tudor Thomas will read a paper on "Donor material for corneal grafts," and after an interval there will be papers by Mr. George Black, on "Some aspects of the treatment of simple detachment of the retina" and by Miss Savory, of London, on "Some uses of thrombin and fibrinogen in ophthalmic surgery." Tea will be taken in the gardens of Trinity College and the Annual dinner in Keble College Hall.

The second day opens with a paper by Mr. Currie, of Cheltenham, entitled "Eyesight and Industry." The Doyne Memorial Lecture will be delivered by Dr. Leon S. Stone of Yale University; it is entitled "Return of vision and functional polarization in retinae of transplanted eyes." A paper by O. G. Morgan on "Some cases of inflammation of the other eye after cataract extraction" and a symposium of outstanding and instructive cases will be opened by Mr. Frank Law, Mr. H. Campbell Orr, and Mr. Philip Jameson-Evans. Tea will be taken in Keble College Gardens. On July 5 there are papers by Arnold Loewenstein on "Advances in anatomy and physiology obtained by clearing ocular tissues" and by J. P. F. Lloyd on "Making perimetry pay." A ciné film of "a ball and ring implant for use in enucleation," by Norman L. Cutter, of Wilmington, Delaware, is the last item on the programme.

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Third Pan-American Congress of Ophthalmology

DR. TOMAS R. YANES, the President, and the Executive Committee of the Third Pan-American Congress of Ophthalmology have the honour to invite all ophthalmic surgeons to attend the event to be held in Havana, Cuba, from Sunday the 4th to Saturday the 10th, January, 1948.